

Second Edition

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Dr Podcast Scripts for the Final FRCA

EDITED BY | Rebecca Leslie
Emily Johnson
Gary Thomas
Philip Harrington

DRPODCAST

CAMBRIDGE

Medicine

Dr Podcast Scripts for the Final FRCA

‘This book is an extremely valuable resource for anyone preparing for the Final FRCA, and indeed anyone seeking a refresher in clinical anaesthesia. It covers the breadth of subjects required by the exam, with the topics discussed in sufficient detail to give the reader a sound understanding. The question-and-answer format is very engaging and easy to follow. I particularly appreciate the sections in italics where the authors speak to the reader giving context, advice or reminders to guide exam technique and provide support.

As a college tutor, I am grateful to the excellent team of editors and authors for preparing such a superb second edition and I’m looking forward to using it to help the resident doctors I work with to prepare for the Final FRCA, and to refresh my own knowledge too.’

Dr Emma Plunkett
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Dr Podcast Scripts for the Final FRCA

Second Edition

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up-to-date information that is in accord with accepted standards and practice
at the time of publication. Although case histories are drawn from actual
cases, every effort has been made to disguise the identities of the individuals
involved. Nevertheless, the authors, editors, and publishers can make no
warranties that the information contained herein is totally free from error,
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liability for direct or consequential damages resulting from the use of
material contained in this book. Readers are strongly advised to pay careful
attention to information provided by the manufacturer of any drugs or
equipment that they plan to use.

Dedicated to my parents, and to my wife
Georgie for all her support and patience through
my training, exams and work on this book
– Phil Harrington

To Stu and my family – thanks for all your patience
and understanding
– Emily Johnson

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Preface

The second edition of this book has been written entirely by either post-fellowship trainees or recently appointed consultants in anaesthesia and it is intended that it should complement the ever-popular Dr Podcasts. The authors have all had recent experience of anaesthetic examinations or have examining experience and were best placed to write about their areas of interest in an exam-orientated, concise and relevant manner.

The subject areas mirror those of the podcasts and it is hoped that the contents of the book will prove invaluable for candidates preparing for the Final FRCA examinations. The book can either be read in conjunction with the podcasts, or used as a separate resource to aid revision.

The format is built along the lines of a structured oral examination (SOE) and elements of it reflect the composition of model answers to the constructed response questions (CRQs). Key components of the Royal College of Anaesthetists syllabus have been selected for podcasting and publication because they feature regularly in the exam.

Additional topics have been introduced to cover more areas of the syllabus and reflect changes or advances in practice since the publication of the first edition.

Bone, Joint and Connective Tissue

1.1.1 Rheumatoid Arthritis – Jade A Loughran and Sarah F Bell

You are on the ward reviewing a 55-year-old woman who is on your list for a total knee replacement tomorrow. She is keen to tell you that she has severe rheumatoid arthritis that is particularly bad in her hands.

What can you tell me about this condition?

The examiners will be looking for some background medical knowledge to start with.

Rheumatoid arthritis is a systemic chronic inflammatory disease that affects 1–2% of the UK population. It is more prevalent in women, affecting women three times more than men. The onset is generally between 30 and 55 years of age. The exact cause of the condition is unknown, but it is thought to involve an autoimmune process. About 70% of cases are positive for HLA-DR4 and 80% of sufferers are seropositive for rheumatoid factor.

How does the arthritis present?

The patient develops a symmetrical polyarthritis. This may be of varying extent and severity. Rheumatoid arthritis tends to affect the hands, feet, knees, elbows, shoulders and neck.

What is the pathological process that occurs?

The pathological process involves synovitis of joints and tendon sheaths. Loss of articular cartilage and erosion of juxta-articular bone to joint destruction.

What is Still's disease?

Adult-onset Still's disease is similar to the childhood condition of systemic-onset juvenile idiopathic arthritis. It usually presents in young adults with joint pain and inflammation, fever and skin rashes, and can develop into chronic arthritis.

Going back to our case, what might be your concerns regarding anaesthetising a woman with rheumatoid arthritis?

I would want to fully assess the extent of the disease since rheumatoid arthritis is a multisystem condition that can have a number of implications for the anaesthetist. I would be particularly concerned about any airway, respiratory, cardiac, musculoskeletal or haematological problems. I would need to review her drug therapy since this may also influence the anaesthetic technique chosen.

If the woman had not told you that she had been diagnosed with rheumatoid arthritis, what might be some of the symptoms of the condition?

Start with the musculoskeletal symptoms and then move on to the extra-articular symptoms if the examiner will let you.

Rheumatoid arthritis generally presents with **symmetrical** joint problems. Patients experience **pain** and **stiffness** which are worse in the morning and improve with activity. The symptoms may occur as flareups interspersed with good periods. The patient may have noticed progressive **joint deformities**, particularly affecting the hands. The patient may also describe fatigue, weight loss and low mood. About half of sufferers have extra-articular complications, which can involve the airway, respiratory, cardiovascular, neurological, renal and haematological systems.

So, what signs might you observe in the musculoskeletal system?

The patient may have hand or feet signs such as ulnar deviation, boutonniere or swan neck deformities and Z-shaped thumbs. Subcutaneous nodules might be visible.

What would you ask the woman in your history?

I would take a general and a specific history. In the specific history I would be looking to ascertain how and when the rheumatoid arthritis was diagnosed. I would ask which joints were involved and to what extent. In particular I would discuss the range of neck and hand movements. I would also want to know about the drug treatments the patient has tried and is currently taking.

So what can you tell me about rheumatoid neck disease?

The atlantoaxial joint may be affected in rheumatoid arthritis due to erosion of the transverse ligament and breakdown of the odontoid peg. About 25% of patients develop atlantoaxial subluxation. This can lead to acute spinal cord compression or compression of the vertebral arteries. Anterior axial subluxation is the most common type of subluxation and is worsened by neck flexion.

A fixed flexion deformity of the neck may also occur due to fusion of the spine. Concurrent osteoporosis can be further worsened by steroid medication. All of these problems may challenge the anaesthetist since manipulation of the airway may be difficult and should be kept to a minimum.

Let's go back to your history. Is there anything else that you might want to discuss regarding the musculoskeletal system?

I would want to ascertain whether either the temporomandibular or cricoarytenoid joints are affected since this might have implications for intubation and airway management. With regard to the temporomandibular joint I would ask about mouth opening. The symptoms of cricoarytenoid involvement might include dyspnoea, hoarseness, stridor and, rarely, upper airway obstruction.

For a total knee replacement I would consider performing a spinal. I would therefore want to find out whether the patient had had any back involvement or operations. I would also ask whether they would be able to get into a suitable position for this technique to be performed.

What other body systems might you ask about and why?

The examiner is looking to test your knowledge of the multiple complications of this disease. If you list at least some of the systems at the start, you will indicate that you are planning to talk about them and that you have a thorough grasp of the condition.

I would enquire about the respiratory, cardiovascular, haematological, renal and neurological systems. With regard to the respiratory system I would be looking for any evidence of associated pulmonary fibrosis, vasculitis, pulmonary hypertension, pulmonary nodules or pleural effusions. Furthermore, the drugs given for the arthritis might have had unwanted pulmonary effects such as fibrotic changes.

Rheumatoid disease can affect the cardiovascular system in a number of ways. The patient is at an increased risk of arteriosclerosis, myocardial infarction and stroke. Mitral valve disease is present in up to 5% of patients. Pericardial disease such as effusions and inflammation may occur. Cardiac conduction defects may also develop.

The haematological system can be affected by the development of anaemia of chronic disease. Sometimes the platelet count is elevated in association with the generalised inflammatory response during a flareup. A leucopenia may also be seen.

And what about the nervous system?

The patient might develop peripheral neuropathy from rheumatoid arthritis or the drugs given to modify the condition. It is important to discuss and document any neurological changes, especially if a regional or neuraxial block is considered, or if the cervical spine needs to be manipulated.

You mentioned the renal system. Can you tell me anything about the changes that might occur?

The patient may develop renal amyloid or a vasculopathy from rheumatoid arthritis. This might be identified as acute or chronic renal failure. Many of the medications used to treat rheumatoid arthritis may also affect renal function.

Can rheumatoid arthritis affect the liver?

Yes. Felty's syndrome occurs when the inflammatory mediators associated with rheumatoid arthritis cause nodular hepatocyte enlargement. This can be associated with splenomegaly and leucopenia. It affects 1–3% of patients with rheumatoid arthritis.

What about the eyes and skin?

The sclera may be involved. Episcleritis is a feature of rheumatoid arthritis, as is dry eyes. It is therefore important to protect the eyes during general anaesthesia to reduce the risk of corneal ulceration or abrasion. With regard to the skin, rheumatoid nodules are

common. Steroid treatment can cause thin, papery skin to develop, which needs to be handled extremely carefully to avoid trauma.

Let's consider the drugs this patient might be taking. Can you suggest any drugs and their unwanted side effects?

Remember not to forget painkillers and the immunosuppressant or disease-modifying agents.

The patient might be taking regular nonsteroidal anti-inflammatory medications for pain relief. These can cause renal impairment, gastrointestinal ulceration, reduced platelet function and exacerbation of asthma in susceptible individuals.

The disease-modifying antirheumatic drugs such as methotrexate, sulfasalazine and hydroxychloroquine are commonly used in the treatment of rheumatoid arthritis. Methotrexate, in particular, has significant side effects causing fibrotic changes in the lungs and liver. Azathioprine, gold and penicillamine have also been used in the treatment of rheumatoid disease. These drugs can all cause bone marrow suppression, lung toxicity, liver dysfunction, thrombocytopenia, anaemia and renal side effects.

Immune-modulating therapies such as rituximab are increasingly used in these patients. These medications can affect the respiratory, renal, hepatic and cardiac systems and affect the haematological and immune functions.

Steroids are frequently given to patients with rheumatoid arthritis flareups. They have many side effects, including hypertension, diabetes, obesity, adrenal suppression, fragile skin, peptic ulcer disease and electrolyte changes. Patients on high doses or long-term steroids may need additional steroid cover perioperatively.

Having ascertained that this woman has developed hypertension and diabetes since taking regular steroid medication, you find that there are no other extra-articular features of rheumatoid disease. What further information would you want to know in your general history?

I would enquire about previous anaesthetics and whether she has a family history of any problems with an anaesthetic. I would then ask about other medical conditions and go stepwise through the body systems. I would particularly focus on the blood pressure, diabetic control and the cardiovascular history. I would then talk about the medications she was taking and ask whether she had any allergies. Finally, I would discuss starvation history and ask about dentition.

What examination would you perform on this woman?

I would examine the cardiorespiratory, neurological and musculoskeletal systems. I would include a thorough airway assessment in my respiratory examination. I would also look at the condition of the patient's skin and assess their ability to use a patient-controlled anaesthesia (PCA) (i.e. if the disease causes significant pain or deformity of the hands).

How would you investigate this patient prior to surgery?

I would want to review blood tests and an ECG as a minimum. With regard to the blood tests I would check the full blood count, renal function, electrolytes, liver function and ensure that a valid group and save were available. If I had identified features of

respiratory disease in my history or examination, I might request a chest X-ray or pulmonary function tests. If I suspected the rheumatoid disease might be affecting the neck, I would consider performing a neck X-ray in flexion and extension. If these were abnormal, an MRI may be required. With regard to the airway, indirect laryngoscopy might be required if I suspected that the cryco-arytenoid movement was impaired.

From your thorough assessment, you have found that this 55-year-old woman has rheumatoid arthritis that appears to mainly affect her hands and knees. She has hypertension and diabetes. She takes 20 mg prednisolone daily along with paracetamol and diclofenac when her arthritis is particularly bad. Her skin appears fragile.

What would be your preferred anaesthetic technique for this patient?

Try and be decisive about what you would want to do for this patient. The examiner wants to see that you have been in this position before and that you are confident of your abilities.

I would consider performing a spinal anaesthetic with a local anaesthetic and intrathecal opioid. In addition I would consider performing nerve blocks to provide additional postoperative analgesia. I would offer the patient intraoperative sedation. I would use a multimodal approach to postoperative analgesia, including regular paracetamol, non-steroidals, opioids and regular long-acting oral morphine and a short-acting preparation for breakthrough pain.

What are the potential problems with these techniques?

The examiner is not trying to catch you out. They want to know that you can appreciate that there are pros and cons to every anaesthetic.

The spinal might be difficult to perform due to a number of factors. These include problems with patient positioning and altered anatomical landmarks. There is also a potential increased risk of infection so aseptic technique is vital. The spinal with opioid poses the following risks: nerve damage, postdural puncture headache, postoperative nausea and vomiting, respiratory depression and urinary retention. It is important that the nurses looking after the patient postoperatively are aware of these potential complications.

With regard to the peripheral nerve blocks, these may also be challenging because of altered anatomical landmarks. I would use both a peripheral nerve stimulator and ultrasound to aid location of the nerves. Further risks include nerve damage, block failure, intravascular injection and local anaesthetic toxicity.

What other anaesthetic techniques might be appropriate for this case?

The patient could have a general anaesthetic with peripheral nerve blockade and/or morphine PCA. The general anaesthetic will require consideration of the need for intubation. The risk of aspiration should be weighed up against the potential difficulty of intubation and the risks associated with manipulation of the neck. There are many different ways of achieving an appropriate airway, which include an awake fiberoptic intubation, gas induction, IV induction, rapid sequence induction and insertion of either an endotracheal tube or an LMA.

What are the options for analgesia for total knee replacement?

A multimodal approach would be appropriate for patients undergoing total knee replacement. Pre-emptive analgesia can be considered on the day of surgery, which

may include an anti-inflammatory, modified-release opioid and/or neuropathic agents such as gabapentin or pregabalin. Pre-emptive anti-inflammatories have been shown to reduce postoperative opioid requirements. Particular caution should be taken in the elderly when using opioids or antineuropathic agents.

The postoperative analgesia strategy would depend partly on the anaesthetic technique. If a spinal is being performed, intrathecal opioid can be used. Regional nerve blocks could be used with either a general anaesthetic or neuraxial technique, and wound catheters can be used. These techniques allow reduction in opioid requirements. In particular, an adductor canal block allows early postoperative mobilisation when compared with femoral nerve blocks, which can cause quadriceps weakness, or sciatic nerve block, which can cause foot drop, both of which can impact postoperative mobilisation.

Postoperatively I would prescribe regular paracetamol and a nonsteroidal anti-inflammatory unless contraindicated. I would add a long-acting opioid if the patient did not have a PCA, with a short-acting opioid for breakthrough pain. Other adjuncts such as pregabalin/gabapentin would depend on the individual patient.

How would you manage the steroid cover for this patient?

This question is relevant for any patient on steroids. Try and be as clear as possible.

Long-term steroid therapy suppresses the hypothalamic–pituitary–adrenal axis, occurring with doses of 5 mg or more of prednisolone when taken for at least 1 month. This axis is activated by major stress. It is therefore important to consider steroid replacement therapy for patients presenting for surgery to avoid perioperative haemodynamic instability due to adrenal crisis.

The AoA, Royal College of Physicians and the Society for Endocrinology released joint guidelines in 2020 on perioperative steroid cover. They suggest that for patients with adrenal insufficiency of any cause, including long-term steroid therapy, 100 mg hydrocortisone should be given at induction of anaesthesia, followed by a continuous infusion of hydrocortisone 200 mg/24 hours until the patient is able to take oral medications. At this point the steroid can be switched to double their usual oral dose, continued in most cases for 48 hours before dropping back to the usual dose. Major complications or critical illness may confer the need for longer periods of additional cover.

If this patient were taking a different steroid, how would you convert the dose?

10 mg prednisolone is equal to 1.5 mg dexamethasone, 8 mg methylprednisolone and 40 mg hydrocortisone.

Further Reading

Fombon F, Thompson J. Anaesthesia for the adult patient with rheumatoid arthritis. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2006; 6(6): 235–239.

O'Donnell R, Dolan J. Anaesthesia and analgesia for knee joint arthroplasty. *British*

Journal of Anaesthesia Education. 2018; 18(1): 8–15.

Woodcock T, Barker P, Daniel S, Fletcher S, Wass JAH, Tomlinson JW et al. Guidelines for the management of glucocorticoids during the peri-operative period for patients with adrenal insufficiency. *Anaesthesia*. 2020; 75(5): 654–663.

Cardiovascular

1.2.1 Preoperative Assessment and Management of Patients with Cardiac Disease – Timothy JB Wood and Andrew Weir

You are in the pre-assessment clinic seeing a 66-year-old man for a right total hip replacement. The pre-assessment nurse identified a systolic murmur, and the patient suffers from angina and is notably breathless on minimal exertion.

What are the important issues that you would like to explore in the history?

A structured approach is vital, ensuring you mention the routine history so that you don't miss anything out before focussing on the specific cardiac details.

I would introduce myself to the patient and ensure that I am talking to the correct patient and that he is expecting the operation that he is listed for. Then there are a number of general points in the history and some points specific to the cardiac history.

The general history would involve enquiry into:

- Previous general anaesthetics – what were they for and if they presented any problems
- Family history of problems with anaesthetics
- Any regular prescribed or non-prescribed medications
- Any allergies
- Any problems with his gastrointestinal system, particularly heartburn or reflux
- Starvation history
- Any joint problems other than his hips, especially focussing on his cervical spine flexion and extension
- Smoking history
- Alcohol intake

The cardiorespiratory systems are inextricably linked and would form the focus of my attention in this patient.

Specifically, I would enquire about the following:

- Whether he suffers from hypertension, was on treatment for it and how well controlled it is.
- Has he ever had a myocardial infarction, and if so, what treatment did he have?
- Does he suffer from angina, and if so, when and how frequently does it tend to occur, and what does he do when it happens?
- How many pillows does he tend to sleep on and does he become breathless if he sleeps lying flat?

- Does he ever get palpitations or become aware of his heart beating in a funny rhythm? Has he suffered from sudden blackouts or loss of consciousness that have not been explained?
- Has he ever been told that he had a murmur?
- Does he suffer from asthma or chronic obstructive pulmonary disease (COPD) or any other problems with his breathing?
- How much exercise is he able to do before he has to stop, and what is it that limits him? Through specific questioning it should be possible to calculate how many metabolic equivalents (METs) he is capable of.

You mentioned metabolic equivalents. Can you tell me more about them and explain their significance?

Metabolic equivalents provide a means of approximating a patient's ability to increase their oxygen delivery to tissues in response to a physical demand. For example, 1 MET is based on the calculation of the basal oxygen requirement of a 40-year-old man of 70 kg at rest and this equates to 3.5 ml of oxygen per kilogram per minute. By enquiring about what the patient is able to do in their daily activities it is possible to estimate how many METs they can achieve.

- 3 METs is equal to light household work or walking 100 yards on the flat
- 4 METs is equal to climbing 2 flights of stairs
- 6 METs is equal to a short run
- Greater than 10 METs is equal to strenuous exercise.

The significance of this is that less than 4 METs is deemed to represent poor exercise tolerance and this group of patients has a higher rate of perioperative and postoperative cardiovascular and neurological complications. However, often this system is limited due to a patient's medical problems such as arthritis or visual impairment reducing their ability rather than cardiorespiratory problems.

What particular aspects in the history and examination would cause you to be particularly concerned about the murmur?

There are three cardinal features of aortic stenosis:

1. Angina
2. Syncope
3. Dyspnoea.

However, the severity of these symptoms do not correlate well with the degree of the aortic disease. Angina occurs due to the oxygen demand of hypertrophied myocardial muscle outstripping supply. Angina occurs in approximately two thirds of patients with critical aortic stenosis, about 50% of these patients will also have significant coronary artery disease. The precise mechanism of syncope is unclear; however, it would appear that, with a relatively fixed cardiac output, it is not possible to meet the increased demand placed on the cardiovascular system by standing or exercise. Thus, such activities cause a fall in cerebral perfusion and a loss of consciousness. Shortness of breath on exertion, orthopnoea, paroxysmal nocturnal dyspnoea and pulmonary oedema tend to be late symptoms and reflect pulmonary venous hypertension.

On examination, aortic stenosis classically has a slow rising and low volume pulse. However, if aortic regurgitation is occurring simultaneously then the pulse pressure may be increased. A carotid and precordial thrill may be palpated, especially on leaning forward in expiration. The murmur is a harsh late peak systolic murmur heard best at the second right intercostal space. It radiates to the carotids. However, these signs change as the severity of the aortic disease increases and the left ventricle fails, therefore reducing the flow through the valve and the murmur becomes less audible. Therefore, an echocardiograph is required in order to assess the severity of a valve lesion.

You have mentioned echocardiography. How would you interpret the results of this investigation to form a risk level for different grades of aortic stenosis?

Echocardiography can be used to assess the anatomy of the aortic valve, grade the severity and assess the function of the left ventricle. The best indicator of aortic stenosis severity is

- Moderate stenosis is equal to an area of $0.8\text{--}1.2\text{ cm}^2$
- Critical stenosis is equal to an area of less than 0.6 cm^2 .

Occasionally the pressure gradient across the valve is used for grading severity. However, this can be misleading as in high output states such as simultaneous aortic stenosis and regurgitation the severity will be overestimated. More dangerously in low output states where there is a failing left ventricle the flow across the valve will be reduced and so will the gradient, thereby underestimating the disease severity.

Also, the left ventricular function will be graded as normal, mildly, moderately or severely impaired based on the subjective assessment of the echo images.

What blood investigations would you request?

The routine investigations would include:

- Full blood count to exclude any significant anaemia and any platelet or leucocyte abnormality
- Coagulation studies (especially if this patient is on warfarin) and determination of blood group
- Measurement of serum electrolytes, urea and creatinine as these are likely to be disturbed by medication such as diuretics that the patient is taking
- Specific investigations may be required depending on the history, for example these may include liver function tests and B-type natriuretic peptide.

The history is suggestive of severe congestive cardiac failure and angina. How would you investigate this further to decide whether it is safe to proceed to anaesthesia for this patient?

Non-invasive tests could include:

- ECG – looking for any arrhythmia or evidence of ventricular hypertrophy or myocardial ischaemia and infarcts.
- Exercise tests such as the exercise tolerance test – the patient is exercised on a treadmill to a fixed Bruce protocol while ECG readings are taken looking for

ischaemic changes. Alternatively, a simple 6-minute walk test where a patient is asked to walk around a circuit with an oxygen saturation probe attached. The distance achieved over 6 minutes is recorded alongside any desaturation that occurred.

- Cardiopulmonary exercise testing (CPET) – exercise tests are often limited due to disabilities such as arthritis or visual impairment preventing the patient from sustaining exercise. CPEX testing helps overcome this.
- Echocardiography can be used to establish and define the cardiac anatomy and assess ventricular and valvular function; however, this assessment of left ventricular function represents a static measure and gives no indication of the patient's functional reserve. More invasive tests to establish the extent, sites and severity of coronary artery stenosis include coronary angiography.
- Dobutamine stress echocardiogram, which has the advantage of as well as looking at the function of the heart, establishes how well it performs under stress due to the dobutamine.

Can you tell me about cardiopulmonary exercise testing (CPET)?

Cardiopulmonary exercise testing is a means of objective testing to determine a patient's preoperative fitness. It correlates well with postoperative survival and can be used to identify patients who are at increased risk of adverse postoperative outcome for which surgery may be deemed inappropriate, or the patient can be warned of the high risks. It examines the ability of the cardiovascular system to deliver oxygen to tissues during the stress of exercise. This is done by asking the patient to exercise on an ergometer, usually a bike, but the hands can peddle instead if there are difficulties with the lower limbs. At the same time as they are exercising a number of variables are being measured. These are as follows:

- ECG
- Blood pressure
- Expired air flow
- Oxygen uptake from the air
- CO₂ production by the body

From these variables the volume of oxygen consumed (VO₂) in millilitres per minute and the volume of carbon dioxide produced (VCO₂) in millilitres per minute can be calculated. If the VO₂ and VCO₂ are plotted on the same graph against time there is a point where the rise in VCO₂ becomes disproportionate to the rise in VO₂. This indicates the level of exercise where the body has reached its maximum aerobic capacity. This point is termed the anaerobic threshold. An anaerobic threshold of less than 11 ml/kg/min or a VO₂ peak of less than 15 ml/kg/min has been shown to have a higher risk of cardiorespiratory events or death postoperatively. Interestingly, 4 METs equates to about 14 ml/kg/min of oxygen consumption. The advantages of CPET testing are that it objectively quantifies function, as opposed to a subjective assessment of the patient's ability to exercise and perform daily tasks. It provides a means of assessment for patients that fall in the category of not being able to achieve 4 METs or are unable to exercise due to other limitations. An important role of CPET is in planning a patient's postoperative destination. Older and colleagues in their paper in 1999 identified patients at high risk undergoing major abdominal surgery with CPET testing. The patients with anaerobic

thresholds less than 11 ml/kg/min were admitted to intensive care, and invasive monitoring, fluid and inotropes were used to optimise them preoperatively. Mortality in these patients was reduced from 18% to 8.9%.

Are you aware of any risk scoring systems in anaesthesia?

Risk scoring systems can be divided into risk scores and risk prediction models. Risk scores assign weighting to certain risk factors and place patients on a scale, but do not provide an individual risk prediction, whereas risk prediction models use a selection of patient data to provide a specific risk probability for the patient.

The simplest and most commonly used risk score is the American Society of Anesthesiologists' (ASA) scale to assess physical fitness for anaesthesia. This system grades patients from 1, a normal healthy patient, to 5, a moribund patient. The absolute mortality for these grades varies from 0.1% for grade 1 to 9.4% for grade 5.

Another example of an organ specific risk score is Lee's Revised Cardiac Risk Index (RCRI), which uses six risk factors for developing cardiac complications, and helps stratify patients undergoing non-cardiac surgery into different classes of cardiac risk.

The six independent predictors are:

- High-risk surgery (intraperitoneal, intrathoracic, vascular)
- Ischaemic heart disease
- Congestive cardiac failure
- Cerebrovascular disease
- Insulin therapy for diabetes mellitus
- Preoperative creatinine > 176 µmol/l

An RCRI of 0 predicts a cardiac risk of 0.4%, while the presence of >3 risk factors predicts an 11% risk of cardiac complications.

Commonly used risk prediction models include the P-POSSUM score, which uses 12 physiological parameters and 6 operative variables, to provide a predicted 30-day mortality. Newer risk prediction models include the Surgical Outcome Risk Tool (SORT) score which was developed with NCEPOD and uses only six variables, and the NELA score, which is specific to patients undergoing emergency laparotomy. Specific risk prediction models are also available for cardiac surgery. The best known of these is the EuroSCORE.

How could you consider optimising a patient like this for surgery?

Preoperative optimisation could be either medical, surgical or intensive care.

Medical optimisation involves ensuring that any condition that the patient suffers from is being treated optimally. For the cardiovascular system this requires ensuring that ischaemic heart disease and heart failure are treated. If they have suffered a recent myocardial infarction then at least six months should have passed before elective surgery is considered. The patient's coagulation status needs to be considered as they are often on aspirin, clopidogrel or even warfarin because of previous MI, angina, arrhythmia or coronary artery stenting. The indication for these drugs needs to be considered and the risks of stopping them versus the benefits of continuing them during surgery needs to be decided between cardiologist, surgeon, anaesthetist and patient. Arrhythmias should be identified and managed, and heart failure medication should be optimised.

The use of perioperative β -blockade was initially thought to reduce the risk of perioperative myocardial infarction; however, recent research has indicated an increased risk of peri- or postoperative stroke and other adverse events if the β -blockers are started preoperatively. Therefore, current advice is that β -blockers should be continued if the patient is already on them, but should not be started, with the exception of possibly major vascular surgery where the benefits may out-weigh the risks.

Surgical optimisation would include percutaneous coronary intervention, pacemaker insertion or valvular surgery if indicated. Routine coronary revascularisation has not been shown to reduce perioperative risk, and it should only be performed if otherwise indicated, for example, if the patient has suffered a STEMI.

Intensive care optimisation involves the use of fluid and inotropes under the guidance of invasive monitoring to increase the cardiac output and oxygen delivery to tissues of patients who are undergoing high-risk surgery preoperatively and continuing through into the postoperative period. Bland and Shoemaker were the first to use this method in high-risk surgical patients and found a significant reduction in mortality in the treatment group.

Further Reading

Bouch C, Thompson JP. Severity scoring systems in the critically ill. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2008; 8(5): 181–185.

Brown J, Morgan Hughes NJ. Aortic stenosis and non-cardiac surgery. *Supplement Continuing Education in Anaesthesia, Critical Care and Pain*. 2005; 5: 1–4.

Minto G, Biccard B. Assessment of the high-risk perioperative patient. *Supplement*

Continuing Education in Anaesthesia, Critical Care and Pain. 2014; 14(1): 12–17.

Smilowitz NR, Berger JS. Perioperative Cardiovascular Risk Assessment and Management for Noncardiac Surgery: A Review. *Journal of the American Medical Association*. 2020; 324(3): 279–290.

Stones J, Yates D. Clinical risk assessment tools in anaesthesia. *British Journal of Anaesthesia Education*. 2019; 19(2): 47–53.

1.2.2 Arrhythmias – Rebecca A Leslie and Sophia Henderson

Why do arrhythmias occur?

Arrhythmias occur when the normal conducting system in the heart becomes unstable. The three main mechanisms that lead to this instability in the conducting system are:

- Re-entry circuits
- Enhanced automaticity
- Triggered activity.

Tell me a little more about re-entry circuits.

These occur when, in addition to the normal route of conduction via the AV node, there is a second connection between the atria and ventricles. The refractory period in the two connections can be different. This means the action potential will be conducted down one of the connections quicker than the second connection. Subsequently, when the two pathways re-join, the fast connection will have already repolarised, and the refractory period finished, so the action potential from the slower connection is then not only

transmitted onwards through the remaining conduction pathway but also transmitted back up the fast connection (retrograde transmission). The impulse can then enter a repeated cycle of activity, cycling through the two pathways so that it repeatedly activates the atria and the ventricles in rapid succession.

Can you give me an example of an AV re-entry tachycardia?

Wolff-Parkinson-White (WPW) syndrome is an example of an AV re-entry tachycardia.

In this condition, there is an accessory pathway called the bundle of Kent, which conducts more quickly than the AV node. This means that the action potential passing through the bundle of Kent reaches the ventricle more quickly than normal AV action potential. This results in a very short P wave. The ventricle activated by the accessory pathway depolarises slowly, giving rise to the characteristic delta wave on the ECG. When the normal action potential conducted through the AV node and bundle of HIS reaches the ventricle, the rest of the ventricular muscle is depolarised rapidly and the normal QRS complex is produced.

Patients with WPW are susceptible to AV re-entry tachycardias, where there is anterograde conduction through the AV node, and retrograde conduction through the accessory pathway. Occasionally a re-entry tachycardia can occur taking the opposite route, passing anterogradely through the accessory pathway and retrogradely through the AV node. When this occurs only the delta waves are seen on the ECG, as the accessory pathway is activating the whole of the ventricular tissue.

What do you mean by enhanced automaticity?

Almost all cardiac cells undergo slow spontaneous depolarisation (automaticity), leading to the initiation of an action potential. However, the sinoatrial node (SA node) fires at the fastest rate, allowing it, in normal circumstances, to act as the pacemaker.

Spontaneous depolarisation results from a slow increase in the permeability of the pacemaker cell membrane to sodium ions, accompanied by a reduction in potassium permeability (phase 4 in the pacemaker action potential). As a result, the intracellular concentration of sodium ions gradually increases, bringing the membrane potential towards the threshold potential for depolarisation. The rate of firing of the pacemaker cells can be increased by:

- Increasing the rate of rise of phase 4
- Increasing the resting membrane potential
- Lowering the threshold potential.

This can occur as a result of increased sympathetic stimulation, digoxin toxicity, or ischaemia. In some circumstances, the rate of discharge of a group of cardiac cells can become faster than the normal discharge from the SA node, and it will then act as an ectopic pacemaker, for example in atrial flutter.

The final arrhythmogenic mechanism you mentioned was 'triggered activity'. What does this mean?

This refers to a spontaneous action potential, or series of action potentials, which occurs following a normal action potential. There are two different types:

1. Early after-depolarisation
2. Delayed after-depolarisation.

Early after-depolarisation normally occurs in slow resting heart rates and is characterised by an additional depolarisation, which occurs during the plateau phase (phase 2) of the ventricular action potential, or during repolarisation (phase 3) of the myocardium. Normally, activation of Ca^{2+} channels occurs early in the action potential and causes the plateau phase (phase 2) of the myocardial action potential. The inactivation of these Ca^{2+} channels then causes the repolarisation that occurs during phase 3. However, if the cardiac action potential is sufficiently prolonged, these channels can become reset and re-activated, allowing further inward movement of calcium and a second depolarisation of the cell membrane and a second action potential, called an early after-depolarisation. They result in a prolonged QT interval. Delayed after-depolarisation involve an after-depolarisation that occurs after repolarisation is complete during phase 4 of the myocardial action potential. These depolarisations may summate to cause a full action potential, and are thought to be the mechanism behind some sustained tachycardias. They are more likely to occur during faster heart rates and with increased intracellular Ca^{2+} ; their appearance is a feature of cardiac glycoside toxicity.

What factors predispose to arrhythmias?

Remember to classify your answer.

There are several predisposing factors that lead to arrhythmias. These can be classified as those arising from primary cardiac disease and those arising secondary to systemic disease processes.

Predisposing factors arising from primary cardiac disease:

- Myocardial ischaemia especially inferior infarct
- Congenital heart disease
- Excessive vagal tone cardiomyopathies.

Predisposing factors secondary to systemic disease processes:

- Hypoxia
- Hypercarbia
- Hypothermia
- Hypovolaemia
- Electrolyte abnormalities such as hyperkalaemia
- Sepsis
- Pyrexia
- Excessive endogenous catecholamines (pain)
- Excessive exogenous catecholamines
- Thyrotoxicosis
- Tension pneumothorax
- Pulmonary embolism
- Drugs or alcohol withdrawal.

How would you classify arrhythmias?

Arrhythmias can be classified as narrow complex and broad complex arrhythmias. This allows easy recognition and subsequent management.

Narrow complex arrhythmias are those which have a QRS duration of <0.12 seconds, whilst arrhythmias with a QRS duration >0.12 seconds are described as a broad complex

arrhythmia. Narrow complex arrhythmias arise above the bifurcation of the bundle of His. Broad complex arrhythmias usually arise from the ventricles. Occasionally they arise from a supraventricular site and have broad complexes due to aberrant ventricular conduction.

Which different types of narrow complex arrhythmias do you know about?

Narrow complex arrhythmias can arise at various parts of the conducting system:

- In the SA node: sinus arrhythmias, sinus bradycardia or sinus tachycardia
- In the atria: atrial fibrillation, atrial flutter or atrial ectopics
- In the AV node: AV nodal tachycardia
- In the conducting system between the AV node and ventricles: heart block.

Tell me about the different types of heart block.

They can be classified as:

- First-degree heart block
- Second-degree heart block: Mobitz type I and Mobitz type II
- Third-degree (complete) heart block.

First-degree heart block describes a prolonged PR interval >0.2 seconds. Each P wave is followed by a QRS complex. First-degree heart block is normal when it accompanies a vagally induced bradycardia. It is also seen in ischaemic heart disease, hypokalaemia and with the use of digoxin, β -blockers, quinine and some calcium channel blockers. First-degree heart block does not normally progress to other heart blocks and does not normally require treatment. In second-degree Mobitz type I heart block the PR interval increases with each successive beat until one P wave fails to be conducted and is unable to produce a QRS complex. The PR interval then resets to normal and the cycle repeats. It is also known as the Wenckebach phenomenon. Mobitz type I AV block is thought to occur due to abnormal conduction through the AV node. It can occur due to periods of high vagal activity (such as sleep) and a permanent pacemaker is not normally required.

In second-degree Mobitz type II heart block most P waves are followed by a QRS complex, and the PR interval is constant but occasionally there is a P wave that is not followed by a QRS complex. It is thought to occur due to an abnormality in conduction below the AV node, in the bundle of HIS. It can progress to third-degree heart block without warning, so referral to a cardiologist for a pacemaker is required.

In third-degree heart block there is complete interruption of conduction between the atria and the ventricle. As a result, the atria and the ventricles are working entirely independently and the P wave bears no relationship to the QRS complex. A pacemaker is required.

What are the indications for temporary cardiac pacing in the perioperative setting?

Perioperative pacing is required in patients who do not have a permanent pacemaker if they are about to undergo a general anaesthetic and have the following conditions:

- Complete heart block
- Second-degree heart block

- Trifascicular block (left axis deviation, right bundle branch block and first-degree heart block)
- First-degree heart block with left bundle branch block.

Bifascicular block (left axis deviation and right bundle branch block) is not normally a reason for a temporary pacemaker unless the patient has a history of syncope.

Can you tell me some other indications for temporary pacemakers?

Indications for temporary pacing include the following:

- Symptomatic bradycardia that is unresponsive to atropine.
- Myocardial infarction; acute inferior myocardial infarction may damage the artery that supplies the AV node. This can cause complete heart block and bradycardia. Anterior myocardial infarction can involve the bundle branches in the inter-ventricular septum resulting in bradycardia. Pacing will be required if there is second-degree or third-degree heart block.
- Asystole with P wave activity.
- Postoperative period following cardiac surgery, especially in aortic surgery, ventricular septal defect closure, tricuspid surgery and ostium primum repair.
- Some tachyarrhythmias (AV re-entry tachycardia and ventricular tachycardia) can be terminated by 'overdrive' pacing.

Describe ventricular tachycardia (VT).

Ventricular tachycardia is a broad complex tachycardia, defined as a run of at least three consecutive ventricular ectopic beats at a rate of >120 bpm. It arises from either a single focus or multiple foci in the ventricle or from a re-entry circuit. There may be normal capture beats or fusion beats where a normally conducted beat combines with an ectopic beat travelling in the opposite direction.

If adverse signs are present, the patient should undergo DC cardioversion and subsequently an amiodarone infusion should be started to prevent reoccurrence. If the patient is stable a cause should be identified and corrected. If this is unsuccessful then an amiodarone infusion should be started.

What is torsades de pointes?

Torsades de pointes is a specific variant of ventricular tachycardia. It has a classic undulating pattern in the ECG with variation in the size of the QRS complex. It is usually caused by a prolonged QT interval and carries the risk of precipitating ventricular fibrillation and sudden death.

Prolonged QT can be caused by:

- Drugs: quinidine, procainamide, flecainide and tricyclic anti-depressants
- Hypocalcaemia
- Acute myocarditis, especially rheumatoid carditis
- Hereditary syndromes such as Jervell's and Lange-Nielson's syndrome and Romano-Ward's syndrome.

In an emergency, treatment of torsades de pointes involves stopping QT-prolonging drugs, giving β -blockers and magnesium (8 mmol/15 min). Over-ride pacing may be required.

How can a supraventricular tachycardia with aberrant conduction be distinguished from ventricular tachycardia?

Narrow complex tachycardias can combine with abnormal ventricular conduction (left or right bundle branch block) to produce a rhythm that is hard to differentiate from VT.

Features that favour a diagnosis of VT include:

- Concordance between all leads capture beats
- Fusion beats
- Extreme left axis deviation
- AV dissociation
- Failure to respond to intravenous adenosine.

If there is any doubt about the diagnosis, the arrhythmia should be treated as a VT as it has the propensity to progress to ventricular fibrillation.

Describe ventricular fibrillation (VF).

VF is used to describe an ECG that is random and chaotic and has no identifiable QRS complexes. VF can occur after acute myocardial infarction, in electrolyte abnormalities, after electrical shock, or after degeneration of other arrhythmias. It is incompatible with life and requires immediate implementation of the Advanced Life Support protocol. Effective resuscitation relies on prompt delivery of a DC shock, which should be administered as soon as possible and not delayed for intubation or to gain IV access or institute other treatment.

One episode of primary VF (occurring within 48 hours of an infarction) corrected by a DC shock does not require prophylactic treatment. However multiple episodes or secondary VF (occurring after 48 hours of an infarction) should be treated with amiodarone, β -blockers or lidocaine. Implantable cardioverter defibrillators can be placed to deliver low-energy DC shocks in patients who have recurrent episodes of VF.

Further Reading

Arrhythmias and Pathology. Webpage found at: <http://criticalcarenorthampton.com/arrhythmias-and-pathology> (accessed 22 June 2023).

Houghton AR. *Making Sense of the ECG fifth edition*. CRC Press; Taylor & Francis Group, 2019.

Kirkman E. Myocardial action potential. *Anaesthesia and Intensive Care Medicine*. 2006; 7(8): 259–263.

Singh R, Murphy JJ. Electrocardiogram and arrhythmias. *Anaesthesia and Intensive Care Medicine*. 2009; 10(8): 381–384.

Soar J et al. European resuscitation council guidelines 2021. *Resuscitation*. 2021; 161: 115–151.

1.2.3 Hypertension – Matthew JC Thomas and Andrew Weir

You have been asked to see a patient in the preoperative assessment clinic prior to surgery on a colonic tumour. The nurse reports the patient's blood pressure is elevated at 170/90.

This is likely to be a question on the risks of hypertension and appropriate therapy.

What else would you like to know?

I would like to take a full history from this man, including any previous medical history plus medications. I would focus my history to look for any symptoms of cardiac disease or if the patient had known hypertension. I would then perform a full examination and request appropriate tests. I am looking for potential causes of hypertension and also any evidence of end-organ damage.

What causes of hypertension are you looking for?

One of the commonest causes in a hospital clinic is white coat hypertension, which should be excluded. Another common cause is essential hypertension. Prior to diagnosing essential hypertension, it is important to exclude secondary causes of hypertension.

Tell me what you mean by white coat hypertension.

White coat hypertension, which can be induced by both doctors and nurses, is defined as a persistently elevated clinical arterial pressure in combination with a normal ambulatory arterial pressure. It is usually a benign condition. Before a diagnosis of hypertension is made patients should have several blood pressure readings at different times of the day. Obviously, this is not always possible in the hospital clinic but some efforts towards this such as repeated readings following a period of rest are possible.

What are the possible causes of secondary hypertension? Is it common?

Secondary hypertension is rare and is usually identifiable from the history. It can be split into endocrine or renal causes.

Common endocrine causes include the following:

- Cushing's syndrome
- Conn's syndrome
- Hypothyroidism
- Pheochromocytoma

Renal causes include:

- Renal artery stenosis
- Glomerulonephritis
- Polycystic kidneys

If any of these diagnoses are suspected, referral to a specialist clinic would be advised.

Assuming this patient has essential hypertension, you stated that you would look for signs of end-organ damage. What do you mean by this?

In many ways the effects of hypertension on organs is more important than the hypertension itself. The principal effects to concern anaesthetists should be developing heart failure, ischaemic heart disease and renal failure, although other organs such as the eyes can be damaged. These have been associated with an increased incidence of

Table 1.2.3 Lee's Revised Cardiac Risk Index

Criteria	Points
High-risk surgery	1
Coronary artery disease	1
Congestive cardiac failure	1
Cerebrovascular disease	1
Diabetes on insulin	1
Serum creatinine > 177 µmol/L	1
Total	6
Risk of complications (MI, PE, VF, cardiac arrest or complete heart block)	Score
0.4%	0
0.9%	1
6.6%	2
11%	≥3

perioperative complications and form three of the components of Lee's revised cardiac risk index (Table 1.2.3). While not directly linked, hypertension and diabetes are associated, and therefore any evidence of diabetes should be looked for.

How do you identify this damage?

Ischaemic heart disease can be identified based on a history of angina that has hopefully been investigated using exercise tests and possibly angiography. It may also be evident on a preoperative ECG.

Heart failure again can be identified from the history, biomarkers such as a B-natriuretic peptide and imaging such as an echocardiogram.

Renal failure is identified by history but also by elevated urea and creatinine in routine blood tests.

So are you willing to anaesthetise this patient? What is the threshold of blood pressure that you would accept?

Decisions on whether to proceed with surgery should be based on a variety of factors relating to the patient, their condition and the urgency of the operation. There is little doubt that this patient needs blood pressure control, but as this is surgery for cancer I would proceed with the case. The Association of Anaesthetists and British Hypertension Society published guidelines in 2016 which recommended that surgery should be postponed if the blood pressure is greater than 180/110 mmHg.

What problems are associated with hypertension and anaesthesia?

The main risks are that of perioperative myocardial infarction and cerebrovascular disease. These patients often show quite marked cardiovascular lability, and this can be exacerbated by antihypertensive medications. Several authors have shown that the

vasodilatation of anaesthesia will drop the patient's blood pressure to similar levels as a non-hypertensive patient. This puts patients at a risk of relative hypoperfusion of organs such as the kidneys, heart and brain. Recent guidelines recommend that blood pressure should be kept within 10–20% of preoperative baseline values. Certainly, intraoperative hypotension should be avoided.

If this patient were undergoing non-urgent surgery, what would you do?

In that situation if the patient has a blood pressure $>140/90$ mmHg, which is considered to be hypertension requiring treatment by NICE guidelines, it should be confirmed on a minimum of two occasions and lifestyle advice regarding diet and smoking cessation should be given to the patient. If this is unsuccessful drug therapy should be started by their GP.

What drug therapy should be used initially?

NICE recommend an ACE inhibitor if less than 55 years old. If the patient is older than 55 years of age, they recommend a thiazide diuretic or calcium channel blocker.

Further Reading

Hartle A, McCormack T, Carlisle J, et al. The measurement of adult blood pressure and management of hypertension before elective surgery: Joint Guidelines from the Association of Anaesthetists of Great Britain and Ireland and the British Hypertension Society. *Anaesthesia*. 2016; 71 (3): 326–337.

Lee TH, Marcantonio ER, Mangione CM, et al. Derivation and prospective validation of a

simple index for prediction of cardiac risk of major noncardiac surgery. *Circulation*. 1999; 100: 1043–1049.

National Institute for Health and Clinical Excellence. Hypertension in adults: diagnosis and management. www.nice.org.uk/guidance/ng136

Tait A, Howell SJ. Preoperative hypertension: Perioperative implications and management. *British Journal of Anaesthesia Education*. 2021; 21(11): 426–432.

1.2.4 Ischaemic Heart Disease and Congestive Cardiac Failure – Alexander Midgley-Hunt

Ischaemic heart disease (IHD) and congestive cardiac failure (CCF) are widely prevalent conditions with a combined prevalence of approximately 3.2 million people. In 2020, ischaemic heart disease was the 3rd most common cause of death, behind COVID-19 and dementia. It would be expected that a candidate will have a good understanding of both the basic sciences of, and anaesthetic approach to, these conditions.

Ischaemic Heart Disease (IHD)

Please define IHD.

IHD is a series of conditions arising from an imbalance between myocardial oxygen supply and demand due to partial or complete coronary artery obstruction. This manifests as angina or myocardial infarction (MI).

What is the pathophysiology of IHD?

Coronary endothelial damage leads to expression of adhesion molecules. This attracts leukocytes, which deposit on the luminal surface leading to inflammation and lipid plaque formation. Over time this plaque stabilises, developing a fibrotic cap.

Two issues arise from this process. First, progressive expansion of the plaque can narrow the arterial lumen. Second, plaque rupture can expose prothrombotic mediators leading to thrombosis and total or near-total occlusion of the artery. Both scenarios can produce myocardial ischaemia.

What is the significance of IHD during the perioperative period?

Try to avoid falling into the trap of talking only about the ischaemic risks associated with anaesthesia. The examiner is looking to see that you are a thoughtful and holistic perioperative practitioner.

IHD has multifactorial implications for the patient. In the first instance we need to consider the risks of further perioperative myocardial ischaemia, which may occur by two mechanisms.

1. Pain, anaemia, hypothermia and the postoperative stress response may drive tachycardia and hypertension, creating shear stresses and risking plaque rupture. When combined with the prothrombotic and proinflammatory state that accompanies the postoperative period there is significant risk of a further primary myocardial event.
2. Intraoperative hypotension, anaemia, hyperthermia, tachycardia, hypertension or hypoxia creates an oxygen supply–demand imbalance without significant plaque changes. This results in variable myocardial ischaemia with potential for significant hypotension, acute cardiac failure, arrhythmias or sudden cardiac death.

In terms of wide considerations, significant ischaemic heart disease may reduce the patient's functional reserve, placing them at higher risk of non-cardiac complications and limiting their ability to recover after surgery.

The role of the anaesthetist is, first, to assess the patient and ensure that the risks and benefits of the procedure have been considered and discussed before proceeding. Second, it is to avoid intraoperative and postoperative cardiac ischaemia by maintaining myocardial oxygen balance.

Tell me about risk scoring systems in patients with IHD and CCF.

Several scoring systems consider cardiac disease when risk stratifying. These include the ASA grade, P-possum and National Surgical Quality Improvement Program (NSQIP) scoring systems. Lee's Revised Cardiac Risk Index (RCRI), developed in 1999, remains widely used owing to its improved predictive value and ease of use over previous cardiac risk indices.

More recently, the NSQIP-Myocardial Infarction and Cardiac Arrest (NSQIP-MICA) score has been developed. This system provides an individual risk estimate for cardiac arrest and MI based on five variables. It is less user-friendly than the RCRI and does not consider some important postoperative complications such as pulmonary oedema and complete heart block; however, it has a higher predictive value. Therefore, the European Society of Cardiology/ European Society of Anaesthesiology (ESC/ESA) guidelines recommend it as a complementary tool.

Can you give me some more details on the RCRI?

It is worth knowing the Lee's Revised Cardiac Risk Index (RCRI) in detail, as this is one of the most commonly used risk scoring systems.

The RCRI uses six predictive factors to identify patients at increased risk of complications such as MI, pulmonary oedema, ventricular fibrillation, primary cardiac arrest and complete heart block.

These consist of:

1. History of IHD
2. History of cerebrovascular disease
3. History of diabetes requiring insulin
4. Chronic kidney disease
5. History of CCF
6. Undergoing supra-inguinal vascular, intraperitoneal or intrathoracic surgery

The original RCRI data excluded emergency cases and used CK-MB to monitor for myocardial infarction. Studies over the last 15 years, which included emergency cases and used troponin, show that complication rates are likely significantly higher than predicted.

How would you approach the preoperative assessment of the patient with IHD?

I would first determine the urgency of surgery. There may be limited opportunity to preoptimise the emergency patient. In these cases, the decision to proceed with surgery would be on a risk-benefit case, conducted with a consultant present and be fully discussed with the patient.

In the elective setting, my assessment would depend on several factors:

- Surgical procedural risk
- Patient's functional baseline in metabolic equivalents (METS)
- The patient's 30-day cardiac morbidity and mortality – determined by a validated scoring tool such as the RCRI or NSQIP-MICA
- The presence of any unstable cardiac conditions – MI within the last 30 days, unstable angina, acute heart failure, significant arrhythmias or symptomatic valvular disease.

In all cases I would conduct a full anaesthetic history, review the patient's investigations and medications. I would expect all patients with significant IHD to have had a 12-lead ECG within the last 12 months and would perform one if this was not the case.

In patients with low operative and predicted risks (<1%) with stable cardiac conditions, further investigation is unlikely to influence management and it would be appropriate to proceed with surgery. In those with elevated risks or a functional reserve of less than 4 METS, I would look for previous echocardiograms, cardiac biomarkers and non-invasive stress testing (such as stress echocardiography or cardiopulmonary exercise testing). If these were unavailable, then I would consider postponement of the operation whilst arranging some or all of these tests.

Patients with unstable cardiac conditions would require escalation to cardiology to allow for medical optimisation or intervention preoperatively.

In all patients I would consider the most appropriate anaesthetic technique, level of monitoring and postoperative care and discuss the options with the patient, fully explaining the risks and benefits.

What preoperative medications would you expect a patient with IHD to be on?

I would expect a patient to be on antiplatelet agents such as aspirin and clopidogrel, an ACE inhibitor (ACEI) or angiotensin receptor blocker (ARB), a beta-blocker, a statin and a long or short-acting nitrate.

If they were not on these therapies, would you commence them in the perioperative period?

The risks and benefits of commencing perioperative medication differ for each drug. Anti-platelet agents are often stopped to minimise surgical bleeding. Therefore, I would not commence these perioperatively in a stable patient.

ACEIs and ARBs carry significant risk of hypotension and blunt vasopressor responsiveness. ESC/ESA guidelines recommend postponing surgery if possible and starting ACEI at least one week prior to surgery in those with newly diagnosed LV dysfunction.

Whilst patients already on beta blockers should continue perioperatively, starting them *de novo* perioperatively remains controversial. Eight meta-analyses have been conducted on this topic showing variable results. The most influential study to date has been the POISE trial which showed a reduction in non-fatal MI but a 33% increase in all-cause mortality in patients who were commenced on metoprolol perioperatively. This was primarily due to hypotension, bradycardia and stroke. ESC/ESA guidelines advise that beta blockers can be considered in patients undergoing high-risk surgery with an RCRI score of ≥ 2 . Initiation should be low dose and between 2 and 30 days preoperatively, titrated to heart rate and blood pressure.

Statins have a plaque stabilising effect, but are only recommended in patients undergoing vascular surgery and should be initiated at least 2 weeks preoperatively.

How would you manage antiplatelet therapy in the perioperative period?

Patients should continue aspirin for a minimum of 4 weeks after bare metal stenting (BMS) and for 3–12 months after drug-eluting stent (DES) insertion unless there is a high risk of life threatening intraoperative bleeding. Beyond this time aspirin should be continued perioperatively if cardiovascular benefit exceeds bleeding risk.

Patients on dual antiplatelet therapy (DAPT) should continue this for 1 month following BMS, 6 months following DES and 12 months following acute coronary syndrome. This may be shortened to three months as an absolute minimum in patients with a new-generation DES. Surgery within this timeframe should be postponed if possible or bridged with intravenous reversible glycoprotein inhibitors such as tirofiban or eptifibatide.

Beyond this, clopidogrel and ticagrelor should be paused five days prior and prasugrel seven days prior to surgery and restarted as soon as possible, ideally within 48 hours.

What factors influence myocardial oxygen supply?

Myocardial oxygen supply is dependent on three factors, coronary perfusion pressure, perfusion time and arterial delivery of oxygen (DO_2).

Coronary perfusion pressure is determined by the equation:

$$\text{CorPP} = \text{Arterial pressure} - \text{ventricular pressure}$$

Left ventricular end systolic pressure equals arterial pressure; therefore perfusion only occurs in diastole when the arterial diastolic blood pressure exceeds left ventricular end diastolic pressure. The right ventricular, by contrast, is perfused throughout systole and diastole.

As the heart rate increases, diastole is shortened thereby reducing left ventricular perfusion time.

Arterial delivery of oxygen can be determined from the oxygen content equation:

$$\begin{aligned} \text{Oxygen content} = & (1.34 \times \text{haemoglobin concentration (g/L)} \times \text{SpO}_2/100) \\ & + (0.003 \times \text{partial pressure of oxygen}). \end{aligned}$$

What about demand?

Myocardial oxygen demand is determined by heart rate, contractility, and ventricular wall tension.

What would be your choice of induction agents for a patient with IHD and why?

I would induce with a combination of high dose opioids and carefully titrated propofol. Opioids impact sympathetic tone less than agents such as propofol and work as synergistically to reduce the required dose of induction agent creating an overall more cardio-stable induction.

Whilst etomidate could be considered as an alternative, concerns remain around adrenal suppression and I have little experience with using it. Ketamine leads to tachycardia and hypertension, increasing myocardial oxygen demand and risking ischaemia. Therefore, I would avoid both agents.

What are the benefits and drawbacks of neuraxial anaesthesia in IHD?

Neuraxial anaesthesia has many factors to consider. Anticoagulant or antiplatelet agents must be stopped in good time to allow neuraxial anaesthesia. Further, there is the risk of post-block hypotension and potential myocardial ischaemia, particularly if the block rises above T4. On the other hand, with diligent management these drawbacks can be easily managed, and a recent Cochrane review suggested improved 30-day mortality in patients with moderate to severe IHD.

What monitoring would you use?

The level of monitoring should be appropriate to the disease severity and the surgical risk. The primary aim is to maintain myocardial oxygen supply–demand balance. The Association of Anaesthetists guidelines on minimum standards of monitoring are sufficient in most cases.

High-risk patients require further monitoring, including invasive arterial blood pressure monitoring. Cardiac output can be measured non-invasively or minimally using devices such as PiCCO or LiDCO. Routine use of right heart catheters offers no material benefit and is associated with complications.

A central line may be useful if the patient is likely to require inotropic support intraoperatively; however, it is limited as a method of assessing fluid requirements.

ECG monitoring should be conducted throughout. A 12-lead ECG is recommended for high-risk patients if feasible. In those for whom it is not feasible, a CM5 configuration is significantly more sensitive than a standard 3-lead configuration and should be used.

How would you manage an on-table MI?

There is no clear consensus on management of perioperative MI. The management should be approached in an A to E manner, with early escalation for senior support. The core tenets are to minimise oxygen demand, optimise oxygen delivery, commence appropriate investigations and initial management and seek specialist advice.

Intravenous esmolol infusion may benefit patients who are hypertensive and tachycardic to reduce heart rate and oxygen demand. Hypertension can be managed by deepening anaesthesia and ensuring adequate analgesia. Hypotensive patients require judicious fluids, vasopressors and inotropes, ideally directed by echocardiography. Acute arrhythmias may require chemical or electrical cardioversion. Anaemia should be corrected aiming a Hb of 100 g/L. Oxygen should be titrated to saturations of 94–98% and aspirin 300 mg given to all patients.

Investigations required include two 12-lead ECGs 30 minutes apart and serial. ST segment elevation MI is rare but warrants immediate discussion with cardiology and consideration for primary coronary revascularisation. Non-ST segment elevation MI is less likely to benefit from immediate revascularisation but still requires cardiology input.

Surgery should be completed in the most expedient manner and arrangements must be made for postoperative care either in a coronary care unit or high dependency/intensive care.

CCF

What is heart failure and what are the causes?

Heart failure is a condition which occurs when abnormal cardiac structure or function leads to reduced oxygen delivery to tissues at normal filling pressures. It affects approximately one million people in the UK.

The most common causes include ischaemic heart disease, hypertension, alcohol excess, valvular heart disease and idiopathic causes. Rarer causes include viral infection and thyroid disease.

What is the difference between systolic dysfunction and diastolic dysfunction?

Systolic dysfunction is a failure of pumping. Each ventricular contraction leads to a reduced volume of blood ejected. Diastolic dysfunction, by contrast, is a failure of relaxation. Impaired relaxation reduces ventricular cavity size; therefore there is less blood available for ventricular ejection.

How would you manage a patient with CCF intraoperatively?

There is little evidence for choice between regional and general anaesthesia. My general aims would be to preserve cardiac output and myocardial oxygen delivery and minimise myocardial work. I would avoid tachycardia and negative inotropy, and maintain blood pressure and normal sinus rhythm.

In addition to standard anaesthetic care paying attention to temperature, pressure areas and thromboprophylaxis, I would perform a cardio-stable induction with a combination of high-dose opioids and propofol to minimise afterload reduction and would ensure adequate analgesia and depth of anaesthesia intraoperatively. Depth of anaesthesia monitoring could be considered.

I would have vasopressors available but be cautious with their use due to the risks of increasing afterload. I would consider ephedrine in preference to metaraminol owing to its inotropic effects.

Intraoperatively patients should be catheterised to allow close monitoring of fluid balance and as a marker of organ perfusion. All patients undergoing major surgery should have invasive arterial pressure monitoring, targeting the patient’s normal mean arterial pressure +/- 10%, and many would benefit from a form of cardiac output monitoring. The failing ventricle is increasingly reliant on the ‘atrial kick’ for preload, which is lost in arrhythmias; therefore I would aim to maintain sinus rhythm.

How can you assess the severity of CCF?

The NYHA grading system provides a clinical assessment of severity of CCF (Table 1.2.4.1).

Table 1.2.4.1 NYHA grading system

NYHA 1	No symptoms during normal physical activity
NYHA 2	Comfortable at rest, normal physical activity causes symptoms
NYHA 3	Symptoms on minimal exertion, comfortable at rest
NYHA 4	Symptomatic at rest

CCF severity can be more objectively quantified with transthoracic echo, looking at ejection fraction and diastolic dysfunction. In 2022 the British Society of Echocardiography have revised their classification of ejection fraction (Table 1.2.4.2).

Table 1.2.4.2 Ejection fraction

Normal	LV ejection fraction $\geq 55\%$
Borderline low	LV ejection fraction 50–54%
Impaired	LV ejection fraction 36–49%
Severely impaired	LV ejection fraction $\leq 35\%$

Diastolic dysfunction is graded as either normal or grades I–III depending on severity. This is important to note as it is likely to have a bearing on the ease of extubation of the patient following surgery.

Further Reading

Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, et al. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: Executive summary: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014; 130 (24): 2215–2245

Guay J, Choi P, Suresh S, Albert N, Kopp S, Pace NL. Neuraxial blockade for the prevention of postoperative mortality and major morbidity: An overview of Cochrane systematic reviews. *Cochrane Database of Systematic Reviews*. 2014; 2014(1): Cd010108/2014.

Harkness, A, Ring, L, Augustine, DX, Oxborough, D, Robinson, S, Sharma, V,

and the Education Committee of the British Society of Echocardiography. Normal reference intervals for cardiac dimensions and function for use in echocardiographic practice: A guideline from the British Society of Echocardiography. *Echo Research and Practice*. 2020; 7(1): G1–G18.

Kristensen SD, Knuuti J, Saraste A, Anker S, Bøtker HE, Hert SD, et al. 2014 ESC/ESA Guidelines on non-cardiac surgery: Cardiovascular assessment and management: The Joint Task Force on non-cardiac surgery: Cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA). *European Heart Journal*. 2014; 35 (35): 2383–2431.

1.2.5 Postoperative Management of Myocardial Ischaemia – Michael John Scerri and Andrea Binks

A 55-year-old man with a background of hypercholesterolaemia and hypertension is undergoing a knee arthroscopy. He is otherwise a well man, still actively playing social football. During the procedure you notice a change in his ECG, with some ST depression and T wave inversions. Unfortunately, he does not have a preoperative ECG to compare to.

What will be your immediate management of this scenario?

Developing a standard approach to all anaesthetic crises is imperative, deciding quickly on the level of escalation required.

The change in clinical parameter should be considered in context of the patient and where we are in the procedure. I would confirm the reading is true and observe for other changes in heart rate, rhythm, blood pressure or other obvious life-threatening abnormalities. A change in ECG as described requires immediate attention. I would notify the theatre team of the alteration in patient status and apply temporising measures such as administering higher concentrations of inhaled oxygen if arterial saturations are less than 95%, ensure adequate minute ventilation, support a normal blood pressure and attempt to attain a heart rate less than one hundred beats per minute. I would ensure the patient has adequate analgesia and depth of anaesthesia before continuing with a focussed assessment. Consideration will also be given to the need to utilise medications that may offer immediate myocardial protection, such as glyceryl trinitrate or beta blockers.

What are your goals in management for this patient if myocardial ischaemia is suspected?

The overarching goal is to avoid the development of ischaemic myocardium, by balancing myocardial oxygen supply and demand.

This is achieved by a careful management of blood pressure, ensuring adequate diastolic pressure and a suitable heart rate to maintain diastolic filling time and coronary perfusion. This must be balanced against an afterload that is too high, increasing left ventricular wall tension and stroke work and hence myocardial oxygen consumption. The role of prophylactic use of beta blockers is controversial; however, it is important to minimise a rise in heart rate when potential ischaemia is present. It is critical that oxygenation, ventilation and adequate depth of anaesthesia is maintained, with pain and anaemia aggressively treated, as a haematocrit less than 39% has been shown to be an independent predictor of 30-day mortality.

Priority should be given to completing or temporarily abandoning surgery as soon as possible, considering the stage and indication for the procedure and the patient's clinical status. Monitoring may need to be upgraded dependent on the patient's haemodynamics, with consideration for supplemental intravenous access, the placement of an arterial cannula, central venous access and a pulmonary artery catheter to provide tight peri-operative haemodynamic monitoring. Investigations to support the diagnosis of an acute coronary syndrome should be organised and loading with antiplatelets initiated as indicated by local guidelines, cardiologist input and in conjunction with the surgical team after careful consideration of the current bleeding risk. Movement towards definitive reperfusion therapy should occur with haste if it is indicated, and as such a cardiologist should be involved with decision making at the earliest convenience.

How will you proceed with this surgery?

This is a multidisciplinary decision and will be dictated by the indication for the procedure and where in time the procedure is up to. This procedure reflects a non-urgent elective one, and therefore if able the appropriate route of action would be to temporarily abandon it, reducing the surgical stress placed on the patient and expediting a thorough assessment of the patient after emergence. However, if a critical point of the operation has passed or the surgery is emergent, the focus should be on completing the surgery as soon as possible. This may involve calling for surgical assistance or having two consultant surgeons working together to finish in a time-efficient manner.

What factors are associated with perioperative myocardial ischaemia?

There are numerous factors associated with perioperative myocardial ischaemia. Some of these are represented in risk scores such as the NSQUIP calculator and Lee's Revised Cardiac Index, including a history of ischaemic heart disease, heart failure, cerebrovascular accidents, renal dysfunction and diabetes mellitus requiring insulin therapy. Other known risk factors include age greater than seventy, female sex, peripheral artery disease, severe left ventricular dysfunction and emergency surgery. The severity of preoperative angina has been shown to be a predictor of perioperative myocardial infarction, as have large fluctuations in systolic blood pressure, tachycardia and hypothermia. Perioperative myocardial infarctions are also more common to occur in vascular surgery, non-cardiac transplant and thoracic surgery.

How common is perioperative myocardial ischaemia?

It has been estimated to occur in 0.9% of non-cardiac operations performed in patients over the age of 45 years.

What is the pathophysiology of perioperative myocardial infarction?

There are numerous mechanisms that may lead to perioperative myocardial infarction, with the two most common being termed type 1 and type 2 infarctions.

The pathophysiology of type 1 infarctions typically involves a primary coronary event involving large native coronary plaques. These plaques are vulnerable to rupture or fissuring, leading to exposure of thrombogenic material to the coronary circulation, which precipitates acute coronary artery thrombosis. In the perioperative period, this is worsened by the pro-thrombotic state, stress induced hypertension and tachycardia increasing shear forces on the plaque and sympathetically induced vasoconstriction.

Type 2 pathophysiology represents a supply-demand imbalance, in a patient with stable coronary artery disease, likely the more common cause of perioperative myocardial ischaemia. Type 2 ischaemic events are precipitated by factors such as tachycardia, hypotension, fluid overload and increased left ventricular wall tension, anaemia, hypoxia, hypercarbia and stress induced coronary vasoconstriction.

Other types of myocardial infarction have also been defined, but are less important to consider in a general setting. For completeness, however, these include:

- Type 3: Sudden unexpected cardiac death, often with symptoms or signs of myocardial ischaemia
- Type 4a: Myocardial infarction associated with percutaneous intervention
- Type 4b: Myocardial infarction associated with in stent thrombosis
- Type 5: Myocardial infarction associated with coronary artery bypass grafting.

After a team discussion, the surgery is abandoned and the patient emerges from anaesthesia and is moved to the recovery room.

How will you manage the patient in the recovery?

I would formally handover the patient to the recovery staff responsible for the ongoing care of this patient, utilising a structured format, such as SBAR. I would perform a rapid primary assessment, and immediately correct any reversible factors such as hypoxaemia, hypo or hypertension, tachycardia, hypothermia and the presence of pain aggressively.

Once the patient is more alert, I would complete a thorough focussed assessment including a history, examination and ordering of relevant investigations. A standard set of observations and temperature should be measured and serial 12-lead electrocardiographs should be obtained, helping to identify the presence of any dynamic ischaemic changes. An arterial blood gas to assess haemoglobin concentration, electrolytes and serial troponin levels should be organised. At the earliest convenience I would engage the cardiologist physicians, if not already contacted, to help guide patient management.

What is meant by the term MINS, and how is this relevant to diagnosing postoperative myocardial ischaemia?

Diagnoses of myocardial ischaemia can be difficult in the postoperative period, often confounded by persistent sedation and the presence of significant analgesia, which may render the ischaemia asymptomatic. The classical methods of diagnosing myocardial ischaemia by considering ischaemic signs and symptoms coupled with raised biomarkers may not be sensitive.

MINS stands for myocardial injury after non-cardiac surgery, and is defined as any myocardial injury caused by ischaemia, occurring within 30 days of non-cardiac surgery. The VISION study (2017) identified MINS, diagnosed by a raised high sensitivity troponin within 72 hours of non-cardiac surgery, as an independent risk factor for postoperative 30-day mortality. This is regardless of the presence or absence of ischaemic signs or symptoms. Specifically, an absolute number for postoperative high-sensitivity troponins measured at any point over the 72 postoperative hours of greater than 20 ng/L or a rise by 5 ng/L was associated with increased 30-day mortality.

Interestingly the VISION study highlights that 93.1% of patients who experienced MINS did not display a classical ischaemic feature, supporting the difficulties of diagnosing postoperative myocardial ischaemia. MINS therefore enables the diagnosis of myocardial ischaemia in the postoperative setting, by an elevated high-sensitivity troponin assay, in the absence of ischaemic signs or symptoms, provided an alternative non-ischaemic diagnosis is not likely.

What would indicate that this patient requires immediate percutaneous intervention?

This decision is best made by the cardiologist, and as such they should be involved in the patient's care from early in the case. Urgent percutaneous intervention, however, would be indicated in a patient who is in cardiogenic shock, is hemodynamically unstable or shows persistent signs of ischaemia, for example on electrocardiograph or echocardiogram, despite medical therapy.

What medications may be useful in managing an acute coronary syndrome?

Consideration of the pathophysiology of the ischaemia lends itself nicely to the types of pharmacological interventions that may be useful. The pro-thrombotic state in the perioperative period may be combatted by the use of antiplatelet and anticoagulant drugs such as aspirin, clopidogrel, prasugrel and heparin. The decision on the use of

such medications must be made by a multidisciplinary team, balancing the bleeding risk of the surgery with the ischaemic risk to the myocardium, and following local hospital protocol.

Beta blockers may be useful in patients with preserved left ventricular function to reduce heart rate and contractility, thereby reducing myocardial oxygen demand and improving supply. Reduction of blood pressure and contractility will also lead to reduced shear forces in coronary vasculature, reducing shear forces on coronary plaques.

Vasodilators such as glyceryl trinitrate may be useful to promote coronary vasodilation and coronary blood flow, improving symptoms and resolving ECG changes. They need to be utilised with caution, however, not to lower systemic blood pressure too much and subsequently reduce the driving pressure to maintain coronary artery blood flow.

How do patient outcomes differ when comparing those who suffer perioperative myocardial infarction to those who don't?

The presence of perioperative infarction has been linked to increased rates of cardiac arrest and cardiogenic shock, which ultimately leads to greater in-hospital mortality and length of hospital stay. Interestingly, those patients who underwent invasive management (i.e. percutaneous intervention) had lower in-hospital mortality than those managed conservatively, despite the larger incidence of bleeding complications.

Further Reading

Al-Attar N. Postoperative myocardial infarction. An article from the e-journal of the ESC council for Cardiology Practice. 2011; 10(4). Accessed online at Postoperative myocardial infarction (escardio.org).

The Joint Task Force on Non-cardiac Surgery: Cardiovascular Assessment and Management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA). 2014 ESC/ECA Guidelines on non-cardiac surgery: Cardiovascular assessment and management. *European Heart Journal*. 2014; 35: 2383–2431.

Landesberg G, Hillel Z (2015). Chapter 47: Electrocardiography, Perioperative Ischaemia and Myocardial Infarction.

Miller's Anaesthesia. 8th Edition. Elsevier Saunders, Philadelphia USA.

Smilowitz NR, Gupta N, Guo Y, Berger S, Bangalore S. Perioperative acute myocardial infarction associated with non-cardiac surgery. *European Heart Journal*. 2017; 38: 2409–2417.

Thygesen K. What's new in the fourth universal definition of myocardial infarction? *European Heart Journal*. 2018; 39: 3757–3765.

Writing Committee for the VISION Study Investigators. Association of Postoperative High-Sensitivity Troponin Levels with Myocardial Injury and 30-Day Mortality among patients undergoing Noncardiac Surgery. *Journal of the American Medical Association*. 2017; 317(16): 1642–1651.

1.2.6 Valvular Defects – Nirav D Patel and Mari H Roberts

This topic is likely to come up as a physiology SOE or a clinical long case. In the clinical long case, you will have time to plan your answer. Try and focus on preoperative assessment, outstanding investigations, how these investigations would affect your anaesthetic management and postoperative care. When it comes to the anaesthetic plan remember to mention monitoring, induction, maintenance and emergence of anaesthesia.

You are asked to anaesthetise an 86-year-old man for a dynamic hip screw insertion. He unexpectedly collapsed getting out of bed and fractured his hip. He has no significant past medical history and does not take any regular medication. On admission, the accident and emergency foundation doctor identified a systolic murmur over the apex of the heart and has requested a transthoracic echocardiogram. How would you assess the patient's fitness for anaesthesia?

If this is a long case, you will be given time to prepare for your answers. It is good practice to summarise the information available to you prior to answering any questions.

In summary, this is an 86-year-old man presenting for emergency surgery procedure with likely undiagnosed valvular disease, possibly aortic stenosis, that may have been a causative factor in his presenting complaint.

I would start by seeing the patient on the ward for a pre-anaesthetic assessment. I would review any medical notes if available and take a detailed history and perform an examination. Focus of my assessment would be to ascertain his general fitness, exercise tolerance and what symptoms may limit his exercise. In addition to this I would specifically ask about any cardiac and respiratory symptoms such as angina, syncope, dyspnoea, palpitations, orthopnoea, and nocturnal dyspnoea to assess the severity of his likely valvular disease.

This man says he rarely gets out of the house due to frequent dizzy spells. He has help with cleaning, manages his own cooking but is unable to climb a flight of stairs without having a rest halfway.

What do you think of his functional capacity?

He has poor functional capacity, less than 4 METS. It sounds like he has pre-syncopal episodes prior to his fall which could be caused by stenotic valvular disease.

What signs are you looking for on examination?

The pulse would be checked for rate, rhythm, and character. He may be in atrial fibrillation and have a slow rising pulse indicating aortic stenosis. On auscultation there may be an ejection systolic murmur at the apex of the heart or second right intercostal space. The murmur may radiate to the carotids. There may be signs of fluid retention such as bibasal crackles, sacral and pedal oedema.

What investigations would you ask for and what would you be looking for?

I would ask for a full blood count, renal profile, ECG and await the echocardiogram report. In the blood tests I would check the patient's haemoglobin to see if he was anaemic, creatinine to assess his renal function and electrolytes to check for salt imbalances such as hypokalaemia. I would look for signs of aortic stenosis on his ECG such as arrhythmias, left ventricular hypertrophy, t-wave inversion and ST segment depression indicating left ventricular strain. A chest X-ray may show left ventricular hypertrophy; however, this may only be present when left ventricular failure occurs.

On the Echocardiogram report I would look at the aortic valve area, pressure gradients across the aortic valve, left and right ventricular function and structure and for atrial dilatation.

If the echocardiogram cannot be done for a further 48 hours, would you delay the surgery?

Ideally, it would be good to have the results of the echocardiogram to confirm the diagnosis of aortic stenosis as it may affect the choice of anaesthetic given; however, national guidance on hip fracture management recommends operating and fixing the fracture within 48 hours of injury. By delaying the surgery, the patient is at more risk of complications of being immobile such as chest infections. The decision on timing of surgery should involve an early multidisciplinary team approach between the patient, family, anaesthetist, surgeon, cardiologist and orthogeriatricians. Due to the significant risk of morbidity and mortality by delaying surgery, I suspect surgery would proceed and without the echo results. I would manage the patient as if he has severe aortic valve disease with optimisation of preoperative parameters such as haemoglobin, electrolytes, invasive blood pressure monitoring and consideration for higher level of care postoperatively.

It is likely at this point you may be given results of the investigations, especially the echocardiogram.

The echocardiogram shows that the patient has aortic stenosis with an aortic valve area of 0.8 cm^2 and a mean gradient of 45 mmHg. What is the classification of aortic stenosis according to valve area and gradient?

According to British Society of Echocardiography guidance, a valve area of $1.6\text{--}2.5 \text{ cm}^2$ is mild stenosis, $1\text{--}1.5 \text{ cm}^2$ is moderate, less than 1 cm^2 severe and less than 0.7 cm^2 is very severe. The mean pressure gradient across the valve can also be used to assess severity – a pressure of less than 20 mmHg is mild, 20–39 mmHg is moderate, 40–59 mmHg is severe and greater than 60 mmHg very severe. This man has severe aortic stenosis.

What are the causes of aortic stenosis?

Degenerative calcified aortic stenosis is the most common cause in the United Kingdom, followed by bicuspid aortic valve. Worldwide rheumatic disease is the commonest cause of mixed aortic and mitral valve disease. In degenerative calcified aortic valve disease, there is mechanical stress over time causing fibrosis and calcification of the valve and it occurs in the elderly.

What are the pathophysiological changes that occur as the valve area decreases?

As the severity of aortic stenosis progresses there is increased left ventricular outflow obstruction. To maintain the pressure gradient across the valve, the left ventricle hypertrophies. As the valve becomes more stenosed, the ventricle becomes stiff and less compliant causing diastolic failure due to impaired relaxation and filling. The impaired

diastole means ventricular filling is dependent on atrial contraction and the cardiac output becomes fixed. According to Ohms law, maintenance of blood pressure is reliant on systemic vascular resistance. Left ventricular hypertrophy and the increase in after-load increases ventricular oxygen requirements. However, due to the low aortic pressure and increased left ventricular diastolic pressure, myocardial oxygen supply is reduced. This mismatch between supply and demand of myocardial oxygen causes ischaemia and symptoms of angina present. Eventually the contractility of the ventricle will begin to fail causing a reduction in cardiac output and reduced mean pressure gradient across the valve.

Moving on, after making your assessment of the patient and reviewing the investigations, tell me how you would anaesthetise this patient. What are your specific concerns with regards to his aortic stenosis?

My anaesthetic plan for this patient would be for general anaesthesia. This patient is high-risk and should have consultant level surgical and anaesthetic input and an experienced anaesthetic assistant.

Prior to bringing up the patient to theatre I would make sure haemoglobin is optimised and blood is available in case of significant blood loss and maintenance IVI is running to avoid dehydration. I would have mandatory monitoring as well as invasive arterial blood pressure prior to induction of general anaesthetic to manage any changes in blood pressure promptly and EEG monitoring to titrate the anaesthetic. I would place two venous cannula – one of which is wide-bore for blood products, the other for a phenylephrine or metaraminol infusion to maintain the patient's blood pressure at induction and intraoperatively. Adequate patient temperature monitoring and warming will be set up and used also. For intra and postoperative analgesia I will place a fascia iliaca block to minimise opiate use in the perioperative period.

After preparing the monitoring and emergency drugs, I would induce the patient in theatre to minimise disconnection of monitoring and lines. Induction of anaesthesia would be with 1–2 mcg/kg of fentanyl, commencing the vasopressor infusion and very slow titration of propofol or gas induction with sevoflurane. Once the patient is asleep, I would administer muscle relaxation and secure the airway with an oral endotracheal tube. Volatile anaesthesia would be maintained according to EEG monitoring and I would ensure blood pressure is maintained to preoperative levels during the procedure. For antiemetic I would give ondansetron and analgesia, some paracetamol and further small aliquots of fentanyl. I would avoid NSAIDs in the elderly population due to the risk of kidney injury exacerbated by pre-op dehydration. Regular haemoglobin checks can be made during the procedure from the arterial line. If the haemoglobin falls below 90 g/L, I will transfuse the patient.

At the end of the case, the patient will be extubated once they are obeying commands and transferred to recovery with oxygen, routine, and invasive arterial monitoring. I would keep the patient in recovery for an extended period to ensure adequate pain relief, stable blood pressure and recovery from anaesthetic. The continuation of vasopressors may be required to maintain a suitable blood pressure. The patient's haemoglobin should be checked, and transfusion given if haemoglobin is less than 90 g/L. After recovery, this patient would benefit from further observations on high dependency unit or post-anaesthesia care unit.

What haemodynamic goals are you aiming for and how would you maintain them?

Categorise this answer into systemic vascular resistance, heart rate, rhythm, preload, and contractility.

Firstly, I would look to support systemic vascular resistance and diastolic blood pressure to maintain coronary perfusion. The use of arterial line blood pressure monitoring would help detect changes in blood pressure so that hypotension can be managed quickly, and the use of a vasopressor infusion can maintain blood pressure at preoperative levels.

In terms of heart rate my goals would be to maintain low-normal heart rate of around 60 beats per minute. Tachycardia should be avoided as it will increase myocardial oxygen consumption. In addition to this, coronary perfusion occurs during diastole and tachycardia will reduce diastolic time. Severe bradycardia should also be avoided as it can reduce the cardiac output and coronary perfusion. The use of fentanyl will reduce the sympathetic response at intubation and a regional technique for analgesia will help reduce the surgical stress of the operation. It is important to maintain sinus rhythm as ventricular filling is depending on atrial contraction. I would ensure electrolyte levels are within normal range perioperatively.

Pre-load should be maintained by preventing dehydration preoperatively and giving IV fluids. Vasodilation should be avoided as it will suddenly reduce venous return, hence why spinal anaesthesia should be avoided in cases of severe aortic stenosis.

Contractility of the myocardium should be maintained, and careful titration of anaesthetic agents is paramount and can be guided by EEG monitoring. Ischaemia should also be avoided as this will reduce contractility.

Ok, let's move on to other valvular lesions starting with mitral stenosis.

Knowledge of other common valvular lesions is important as they can easily come up as a long case or physiology SOE.

What are the common causes of mitral stenosis?

The commonest cause of mitral stenosis is rheumatic heart disease followed by degenerative calcification. Rarer causes include radiation, carcinoid disease and congenital heart disease.

How is mitral valve stenosis classified?

Mitral stenosis can be classified according to valve area and mean gradient. A valve area of less than 1 cm^2 and gradient of greater than 10 mmHg are indicative of severe mitral stenosis.

What is the pathophysiology as the stenosis worsens?

Symptoms of mitral stenosis occur relatively early compared to aortic stenosis and a valve area of less than 2.5 cm^2 is usually clinically significant.

As the valve becomes more stenosed, passive filling becomes more difficult and there is more reliance on atrial contraction. This causes dilatation of the left atrium. As the

disease progresses there is increase in left atrial pressure and pulmonary hypertension. Atrial fibrillation is also common.

What are the haemodynamic goals when anaesthetising a patient with mitral stenosis?

Like aortic stenosis, mitral stenosis results in a fixed cardiac output state and reliance on systemic vascular resistance.

Systemic vascular resistance should be maintained and heart rate low-normal at 60 beats per minute. Maintenance of sinus rhythm is imperative. Maintenance of normovolemia and careful titration of preload is needed. Reduction in venous return can reduce left atrial pressure and reduce ventricular filling. However, sudden increases of venous return should also be avoided as this can cause pulmonary oedema. Contractility should also be maintained, and reduction should be avoided.

In the presence of pulmonary hypertension, hypoxaemia, hypercarbia, acidosis, nitrous oxide and sympathetic stimulation should be avoided as it will increase pulmonary vascular resistance and acute right ventricular failure may be precipitated.

Now we will move onto regurgitant lesions. What are the causes of mitral regurgitation?

Mitral regurgitation can be classified into primary or secondary. Primary is due to pathology of the valve itself that prevents normal closure such as rheumatic disease or leaflet prolapse. Secondary is caused by left ventricular dysfunction such as dilatation of the left ventricle. Causes can also be split into acute, for example papillary muscle rupture secondary to MI, and chronic, secondary to valve degeneration.

What is the pathophysiology of mitral regurgitation?

During ventricular contraction, there is back flow of blood into the left atrium causing volume overload of the left atrium. In chronic mitral regurgitation, the left atrium dilates in response to volume overload. The left ventricle also dilates increasing end diastolic volume. This is initially well tolerated and ventricular function preserved as blood is pumped through the aortic and mitral valve but as the ventricle dilates further, contractility is decreased, and left ventricular systolic function decreases. Atrial fibrillation is also common in these patients; however, it is better tolerated than in stenotic lesions as passive filling is maintained.

Acute mitral regurgitation is less well tolerated, for example after papillary muscle rupture following MI or endocarditis. There is no time for compensatory mechanisms as there is an acute increase in left atrial and ventricular volume and pressure. This results in a reduction in cardiac output, left ventricular failure, pulmonary oedema and right ventricular failure due to increased right ventricular strain.

How do you classify mitral regurgitation?

The classification of mitral regurgitation can be made by various parameters in echocardiography. A regurgitant fraction of less than 30% is considered mild and greater than 50% severe. A left ventricular ejection fraction of less than 60% indicates significant systolic dysfunction.

What are the haemodynamic goals in a patient with mitral regurgitation undergoing anaesthesia?

The haemodynamic goals for regurgitant lesions are to maintain forward flow to reduce the regurgitant lesion.

Systemic vascular resistance should be low to promote forward flow, therefore sharp increases in systemic vascular resistance should be avoided. Vasoconstrictors should be used with caution.

Heart rate should be maintained at a higher rate, 80–100 to reduce filling time of the left ventricle and reduce systolic duration, which will decrease the regurgitant volume. Sinus rhythm is preferred; however, passive filling is maintained in mitral regurgitation and atrial fibrillation is better tolerated than in stenotic disease.

Preload should be maintained, but sudden increases avoided due to the risk of pulmonary oedema. Contractility should also be maintained. In patients with acute mitral regurgitation and left ventricular failure the use of inotropes and ionodilators may be required.

In patients with pulmonary hypertension, further increases in pulmonary vascular resistance should be prevented by avoiding acidosis, hypoxia, hypercarbia, and nitrous oxide.

What are the causes of aortic regurgitation?

Aortic regurgitation or incompetence can be classified into acute causes such as aortic dissection and bacterial endocarditis, or chronic, such as rheumatic disease, syphilis and connective tissue diseases such as Marfan's disease.

What is the pathophysiology of chronic aortic regurgitation?

During diastole, a significant proportion of the stroke volume returns into the left ventricle. This causes an increase in volume load in the left ventricle. Initially the ventricle will dilate and increase ventricular contraction as per the Frank–Starling relationship, where the increase in myocardial fibre length, increases the force of myocardial contraction. As the ventricle further dilates over time, the fibres will stretch too much and contractility falls.

What are the haemodynamic goals when anaesthetising a patient with aortic regurgitation?

Similar to mitral regurgitation, the aim is to maintain forward flow, reduce regurgitant flow and reduce ventricular distension.

Systemic vascular resistance is kept low to reduce ventricular outflow resistance, promote forward flow of blood and maintain cardiac output.

Heart rate should be high normal (80–100 beats per minute) to reduce time for regurgitant flow and prevent distension of the ventricle. Sinus rhythm should be preserved.

Preload should be maintained with adequate volume loading to ensure the dilated ventricle is well filled. Contractility should be maintained as with other valvular lesions.

Further Reading

Brown J, Morgan Hughes NJ. Aortic stenosis and non-cardiac surgery. *Continuing Education in Anaesthesia Critical Care and Pain*. 2005; 5(1): 1–4.

Guideline by the association of anaesthetists. Guideline for the management of hip fractures 2020. *Anaesthesia*. 2021; 76: 225–237.

Holmes K, Gibbison B, Vohra HA. Mitral valve and mitral valve disease. *British*

Journal of Anaesthesia Education. 2017; 17 (1): 1–9.

Ring L et al. Echocardiographic assessment of aortic stenosis: A practical guideline from the British Society of Echocardiography. *Echo Research and Practice*. 2021; 8(1): G19–G59.

Robinson S et al. The assessment of mitral valve disease: A guideline from the British Society of Echocardiography. *Echo Research and Practice*. 2021; 8 (1): G87–G136.

1.2.7 Cardiomyopathy – Richard LI Skone and Andrew Weir

This topic, as with many, may start in very broad terms. Be prepared to have a simple classification ready. We have discussed the four main groups of cardiomyopathy here, and peripartum cardiomyopathy is mentioned as an important subgroup.

Tell me what you know about cardiomyopathies.

A cardiomyopathy is a disease of the heart muscle. Although the World Health Organisation (WHO) defines them as being cardiac muscle diseases without an apparent precipitating cause, classically they have been described as being idiopathic or secondary to other disease processes, and they affect 1 in 500 people in the UK.

The main categories of cardiomyopathies according to the 2008 European Society of Cardiology Working Group on myocardial and pericardial diseases are as follows:

- Dilated cardiomyopathy
- Hypertrophic cardiomyopathy
- Restrictive cardiomyopathy
- Arrhythmogenic right ventricular cardiomyopathy.

Although they vary in their pathophysiology, any of the cardiomyopathies can present with arrhythmias and/or heart failure.

The examiner may then ask you to focus on one particular type of cardiomyopathy.

Tell me about dilated cardiomyopathy (DCM).

DCM has a prevalence of 1:2500 people. It is characterised by ventricular chamber enlargement, normal left ventricle (LV) wall thickness and systolic dysfunction. This leads to a progressive decrease in effective contractile function of the myocardium and consequently, heart failure.

The sequelae of DCM include:

- Heart failure
- Arrhythmias
- Embolic events.

The low cardiac output state also leads to activation of the renin–angiotensin system. Eventually DCM leads to heart failure or sudden cardiac death. DCM has a familial cause in 20–30% of cases.

It can also be caused by the following:

- Alcohol
- Infection – Coxsackie virus or HIV
- High output states – pregnancy, thyrotoxicosis, anaemia
- Drugs – heavy metals, cocaine
- Thiamine deficiency
- Pheochromocytoma.

The prognosis of DCM is quite poor. The Framingham study showed that life expectancy is inversely proportional to the severity of the disease at presentation. The 5-year survival rate is between 40 and 50% once heart failure has been diagnosed. Medical management includes treatment of heart failure including ACE inhibitors, beta blockade, diuretic therapy and anticoagulation. Non-medical management may include ventricular assist devices, ICDs or transplantation.

Tell me about hypertrophic cardiomyopathy.

Hypertrophic cardiomyopathy is primary myocardial hypertrophy of the left ventricle, in the absence of other abnormalities. It is the most common purely genetic cardiovascular disease. It has a prevalence of approximately 1:500 people with autosomal dominant inheritance, and 60% of unexplained LV hypertrophy is caused by HCM. It carries a mortality rate of 1% per year and is the most common cause of sudden cardiac death in young people.

It causes ventricular hypertrophy with preserved systolic function, poor ventricular compliance and diastolic dysfunction. Hypertrophy of the septum with outflow obstruction occurs in approximately one third at rest, and one third during provocation.

The disease can be asymptomatic or can present in a range of ways including with dysrhythmias, angina, heart failure or sudden death.

Familial hypertrophic cardiomyopathy occurs because of defects in the sarcomeric proteins. This in turn leads to myofibril disarray and fibrosis (even in seemingly unaffected areas). These areas can be pro-arrhythmogenic and lead to ventricular dysrhythmias.

Myocardial ischaemia can occur because of ventricular wall hypertrophy, elevated diastolic pressures and increased oxygen demand.

The left ventricular outflow obstruction occurs because of inter-ventricular septal hypertrophy. The degree of outflow obstruction is often dynamic and can be affected by the patient's volume status.

Predictors of sudden cardiac death (SCD) in HCM include:

- Previous cardiac arrest
- Family history of SCD
- Episodes of sustained or non-sustained VT
- Syncope
- Abnormal blood pressure responses to exercise.

First line medical management is with beta blockade, which prolong diastolic time and allow for improved left ventricular filling. ICDs are effective in reducing the risk of sudden cardiac death in high-risk patients. Surgical myomectomy can be an option for select patients.

Tell me about restrictive cardiomyopathy (RCM).

RCM is the rarest of the three main cardiomyopathies, and is characterised by ventricular diastolic dysfunction due to fibrotic or infiltrative myocardial changes. Usually, normal systolic function is well preserved.

Its causes can be idiopathic or secondary to infiltrative diseases such as:

- Amyloidosis
- Sarcoidosis
- Haemochromatosis
- Ischaemic heart disease.

Presenting symptoms are usually those of biventricular failure including dyspnoea, oedema and orthopnoea. Echocardiography and cardiac MRI may be useful to differentiate between true RCM and constrictive pericarditis. Medical management is again based on symptomatic treatment of cardiac failure, and PPMs or ICDs for selected patients with conduction abnormalities.

The prognosis for RCM depends largely on its cause. The outlook for patients with RCM secondary to amyloidosis is significantly worse than for those who have well-managed haemochromatosis. Death is usually due to progressive low cardiac output failure.

Tell me about arrhythmogenic right ventricular cardiomyopathy (ARVC).

ARVC is a rare condition in which the right ventricle tissue becomes replaced by adipose and fibrotic tissue. This leads to ventricular arrhythmias, loss of function or SCD. It has a prevalence of 1 in 5000 people with a largely genetic predisposition. It also has a striking incidence in athletes.

Patients usually present in their 20s to early 40s; 80% will present with either syncope, or sudden cardiac death. Other manifestations include palpitations or rarely chest pains.

Sequelae of the fibrotic changes include ventricular tachycardia, a dilated right ventricle (often aneurysmal) and involvement of the right ventricle outflow tract. It progresses to affect the left ventricle in 50% of patients.

Diagnosis is by echocardiography or cardiac MRI and the fulfilling of a certain number of major and minor criteria. Treatment is with implantable cardiac defibrillators, ACE-inhibitors, β -blockers and anticoagulation.

What is peripartum cardiomyopathy?

Peripartum cardiomyopathy is a rare cardiomyopathy presenting in late pregnancy or postpartum. It is usually characterised by a dilated heart and systolic dysfunction. Risk factors include pre-eclampsia, advanced maternal age and multiple pregnancy. The condition carries a high mortality and while more than half will recover cardiac function, many will be left with a chronic cardiomyopathy and heart failure. Important sequelae include thromboembolic disease and arrhythmias, and treatment is comprised of standard heart failure therapy.

The examiner may ask about the anaesthetic management of patients with cardiomyopathies. Again, it is important to remember to give an answer that includes giving a safe and balanced anaesthetic. As well as mentioning the basics of safe anaesthesia it is important to tailor your answer according to the particular cardiomyopathy. The

usual structure of taking a detailed history, examining the patient and arranging investigations still stands. Consider any underlying disease that may have caused the cardiomyopathy. Manage any medications that they may be on such as diuretics or anticoagulants. Remember also that the patients may have implanted cardiac defibrillators.

Further Reading

Elliott P, Andersson B, Arbustini E et al.

Classification of cardiomyopathies:

A position statement from the European society of cardiology working group on myocardial and pericardial diseases.

European Heart Journal. 2008; 29: 270–276.

Honigberg MC, Givertz MM. Peripartum

cardiomyopathy. *British Medical Journal*.

2019; 364: k5287.

Ibrahim RI, Sharma V. Cardiomyopathy

and anaesthesia. *British Journal of Anaesthesia Education*. 2017; 17(11):

363–369.

1.2.8 Pacemakers – Menanta van Velze and William A English

This is an important area of anaesthetic practice that is becoming increasingly complex. The British Heart Rhythm Society published some practical guidance for the management of patients with Cardiac Implantable Electronic Devices (CIED's) in the journal Anaesthesia in 2022 and the examiners will expect a good understanding of the safe perioperative management of these patients.

You are asked to provide a general anaesthetic for a patient with an implanted cardiac electronic device. What would be your preoperative considerations?

All patients for non-emergency surgical procedures should undergo routine pre-admission screening and ideally patients with cardiac devices will be identified and flagged up at this stage already. Many of these patients will have a complex medical history, which will need to be reviewed in detail. Key information specifically related to the cardiac device could be obtained from the patient or their relatives, their pacemaker/device card, clinic letters, their usual cardiology team and from special investigations such as chest X-ray. The key information you would like to know includes:

- The type of device and set mode
- Indication for insertion
- Device manufacturer, model and serial number
- Date of insertion
- Implanting and follow-up hospitals
- Date and results of last check to determine device function and battery life
- Whether the patient is pacemaker dependent.

I would enquire about any symptoms that could suggest pacemaker malfunction, such as dizziness, syncope, dyspnoea, orthopnoea and chest pain.

During my general physical examination, I will aim to elicit signs that could indicate heart failure and establish the exact location of the pulse generator.

I would like to have access to some recent special investigations including:

- A 12-lead ECG
- Serum electrolytes
- A device interrogation by a cardiac physiologist may be required if regular follow-up is overdue
- A chest X-ray (CXR) can also be useful.

What are the different types of cardiac electronic devices that you are aware of and their indications?

A variety of cardiac electronic devices are currently in use and they include:

- Loop recorders used for diagnostic purposes and ECG monitoring
- Permanent pacemakers, which could be single or dual chamber and inserted for symptomatic bradycardias, tachy-brady syndromes and high degree atrioventricular block
- Biventricular pacemakers deliver cardiac resynchronisation therapy (CRT) in patients with heart failure
- Lastly, implantable cardioverter defibrillators (ICD's) are used as primary or secondary prevention in patients who are at high risk of ventricular tachycardias
- A combination of the above can be found in CRT devices with a pacing or defibrillation function.

What would you look for on a Chest X-ray of a patient with a cardiac device coming for surgery?

CXR and ECG interpretation are very often part of your long clinical case, so make sure you have a systematic way of looking at these investigations.

I would interpret the CXR systematically like for any other patient, assessing all the various structures, but specifically looking for signs of cardiac failure.

I would then focus on the cardiac device itself. The number and location of leads, as well as their appearance on the CXR can give you an indication as to which device your patient has. Permanent pacemakers have one or two leads (atrial and/or ventricular), whereas cardiac resynchronisation devices have a third lead going to the coronary sinus. An ICD has a right ventricular lead which contains one or more thick shock coils. It is also important to look for device complications such as lead fracture or migration.

The location of the pulse generator can be determined, and it is often bigger in an ICD than in a pacemaker. Subcutaneous or leadless devices can also be identified.

The manufacturer of the device can also be determined as each company has a specific identifier which is visible on a CXR.

Can you explain the generic pacemaker code?

A generic four-letter code is used to describe pacing modes.

- The first letter indicates the chamber being paced (A = atrium, V = ventricle, D = dual)
- The second letter indicates the chamber being sensed (A = atrium, V = ventricle, D = dual, O = none)

- The third letter indicates the response to sensing (I = inhibited, T = triggered, D = dual)
- The fourth letter indicates the presence or absence of rate modulation (O = none, R = rate modulation)
- A fifth letter can sometimes be present and would indicate the presence or absence of multisite pacing in either the atria, ventricles or both.

The most common modes of newly inserted pacemakers would be VVI, VVIR, DDD or DDIR.

What are your intraoperative considerations and concerns for a patient with a cardiac implantable electronic device?

Minimum monitoring standards, as stipulated by the Association of Anaesthetists, for all patients undergoing general anaesthesia should be adhered to, in particular ECG monitoring, which should be used from the outset. The plethysmograph or palpation for a pulse would provide reassurance of mechanical capture if there is any uncertainty during the procedure. Direct arterial pressure monitoring should be considered for complex or long cases. If a central venous catheter is indicated, great care should be taken during insertion, particularly if the pacemaker has recently been inserted, as the pacing electrodes may become dislodged, leading to pacemaker failure. You also do not want to risk infection of the pacing system. The femoral route should therefore be considered here.

The anaesthetic should be tailored to each patient and care should be taken with the use of suxamethonium, as there is concern that muscle fasciculations may be perceived as intrinsic cardiac impulses, leading to unwanted inhibition of pacemaker function.

Hypoxia, hypercapnia, acidosis and electrolyte disturbances may precipitate arrhythmias. Fluid balance is equally important, as patients with a fixed ventricular rate won't be able to respond to hypovolaemia by raising their heart rate. Homeostasis should therefore be maintained carefully.

Electromagnetic interference (EMI) is a major concern during surgical interventions for these patients and the appropriate actions need to be taken to minimise this. External defibrillation, temporary pacing and cardiopulmonary resuscitation equipment should be available at all times.

What is electromagnetic interference and what are the risks of it?

Electromagnetic interference (EMI) refers to any electromagnetic radiation with the potential to affect implantable devices with the risk of causing inappropriate function. The effects on cardiac devices can vary and may include pacemaker oversensing leading to pacing inhibition, inappropriate anti-tachycardia therapies or shock delivery by an ICD when EMI is incorrectly interpreted as ventricular arrhythmias, or the device can switch to a fixed-rate pacing mode.

Sources of EMI in the surgical environment include surgical diathermy, radiofrequency ablation, electroconvulsive therapy, MRI and other magnetic equipment, transcutaneous nerve stimulation and lithotripsy, amongst others. Even mobile phones or other wireless equipment should not be placed on or near the patient.

How can you minimise EMI and its risk during surgical procedures?

Even though devices are now engineered to be more resistant to EMI, steps should be taken to minimise exposure.

Surgical diathermy is best avoided, but if it has to be used, bipolar is definitely safer than monopolar. It should be used at the minimum energy required, for short bursts of 1–2 seconds at a time, in the cutting, rather than the coagulation mode. The return electrode of the monopolar diathermy should be placed as far away as possible from the cardiac device and in such a position that the pathway from the diathermy electrode to the return plate does not cross or pass near the device or its leads. The use of an ultrasonic cutting device is safe though.

Reprogramming of pacemakers by cardiac physiologists, to an asynchronous mode immediately prior to surgery should be considered, especially in patients who are pacemaker dependent. ICDs should be deactivated in the immediate perioperative period as well. Both temporary magnet deactivation or deactivation by a physiologist are acceptable methods to do so. This will be determined by the urgency of the procedure and the availability of physiologists in your hospital. Rate response functions and sleep modes should also ideally be disabled. The British Heart Rhythm Society provided a very useful table as part of their guidelines on the management of cardiac devices for various procedures in the perioperative period in the journal *Anaesthesia* in 2022, which I always refer back to. They advise that it is reasonable not to deactivate an ICD for surgery below the umbilicus; however, a clinical magnet should immediately be available in case of emergency.

How would you suspect inappropriate pacing inhibition and how would you manage this?

The consequences of pacemaker failure depend upon the underlying cardiac rhythm and thus whether the patient is pacemaker dependent. This highlights the importance of knowing the original indication for insertion of a pacemaker and the results of the last check. ECG monitoring is unreliable to detect pacemaker failure, as the EMI that causes oversensing also interferes with ECG recording. It is therefore important to monitor the plethysmograph trace, arterial line trace (if used) or the patient's pulse.

If pacemaker failure is suspected, EMI should be stopped immediately. If oversensing was the problem, stopping EMI should solve it. If cardiac arrest has occurred and persists, advanced cardiac life support should be provided immediately. External pacing facilities should be available and attempted. Depending on the manufacturer, a clinical magnet can be used to switch the pacemaker into an asynchronous mode.

How would you safely perform external defibrillation or pacing in a patient with a cardiac implanted device?

Defibrillator/pacing pads should be placed at least 10–15 cm away from the edge of the cardiac device. The recommended pad position for a cardiac device located in the left pre-pectoral region, is a right infraclavicular pad with the second pad positioned posteriorly to the left of the spine and just under the scapula. The second pad could also be placed apically if it is far enough from the cardiac device. In any patient with a

deactivated ICD or a pacemaker that has not been reprogrammed to an asynchronous mode, pads should be applied prior to induction of anaesthesia.

You have mentioned magnets as part of the perioperative management of patients with cardiac implantable electronic devices. Tell me more about the role of these magnets.

The magnet I referred to is a clinical or medical grade magnet and is a specifically designed ring or block magnet.

Magnet deactivation is an acceptable and recommended method of ICD deactivation in emergency cases. Most pacemakers would respond with fixed-rate pacing when a magnet is placed over the pulse generator, but this is not completely universal. Leadless pacemakers in particular do not respond to magnets in this way. Asynchronous pacing is usually at a rate of 90–100 beats per minute. Whilst this may be useful in certain situations, leaving a magnet over the pacemaker for prolonged periods is not recommended, as the R on T phenomenon is rare, but possible at this rate.

Inhibition of shock therapy is only temporary and will only be effective during correct magnet placement. It is therefore recommended that the magnet should be secured to the patient's chest with tape for the duration of the procedure. It goes without saying that any ventricular arrhythmias during this time, should be treated by external defibrillation or swift removal of the magnet.

The exact position of the magnet on the device will depend on the manufacturer. Medtronic, Biotronik and Boston supply the majority of ICDs implanted in the UK and they advise positioning of the magnet directly over the device. Abbott (St Jude Medical) on the other hand recommends the magnet is offset inferiorly or superiorly with the curve of the magnet positioned over the top or bottom of the device. Microport (LivaNova) also advises an off-centre position to avoid the header at the top of the device. Biotronik ICDs will revert to normal function after eight hours and the magnet needs to be removed and reapplied if the surgery is to continue longer than this.

Are there any specific postoperative considerations in patients with cardiac devices?

It is the responsibility of the treating clinical team to ensure that all disabled or reprogrammed functions of the device are restored as soon as possible postoperatively by a cardiac physiologist. Any patient with a deactivated ICD should be ECG-monitored with defibrillation pads on until it has been reactivated. If any external shocks have been administered, the device should be checked afterward to confirm correct function. Even though normal cardiac device function will resume on removal of a magnet, it may be good practice and reassuring to do a device check in the postoperative period.

Further Reading

British Heart Rhythm Society. Guideline for the peri-operative management of people with cardiac implantable electronic devices. *Anaesthesia* 2022, 77: 808–817.

Bryant HC, Roberts PR, Diprose P. Perioperative management of patients with cardiac implantable electronic devices. *British Journal of Anaesthesia Education*. 2016; 11: 388–396.

Endocrine and Metabolic

1.3.1 Hormonal and Metabolic Response to Trauma –

Ashley C Davis

A 24-year-old motorcyclist is brought into A&E following a road traffic accident. He has sustained a fracture to his right femur and you are asked to take him to theatre for a femoral nailing.

What is the stress response?

The stress response is a cascade of widespread hormonal and metabolic changes that occur in response to trauma. It is a complex neuroendocrine response, the net effect of which is to increase catabolism and release endogenous fuel stores.

What can trigger the stress response?

The stress response occurs secondary to an exogenous insult such as trauma. It can also occur following surgery, secondary to burns or severe infection and has been reported to occur following strenuous exercise. The magnitude of the response is related to the degree of initial trauma.

How is the response initiated?

Think about local responses, then systemic responses.

At the site of tissue damage, there is a local release of cytokines, which triggers the acute phase response and local inflammation. Somatic and autonomic afferents are transmitted from the point of tissue damage to the central nervous system via ascending pathways in the spinal cord. These signals trigger the hypothalamic–pituitary–adrenal (HPA) axis releasing cortisol, and stimulate the sympathoadrenal response, releasing catecholamines.

What are the characteristics of the resultant state?

The stress response leads to an increase in catabolism, a release of endogenous energy substrates and fluid retention, along with an increase in sympathetic tone. In evolutionary terms this mechanism would help to increase an injured animal's chance of survival. A summary of the response is demonstrated in Figure 1.3.1.

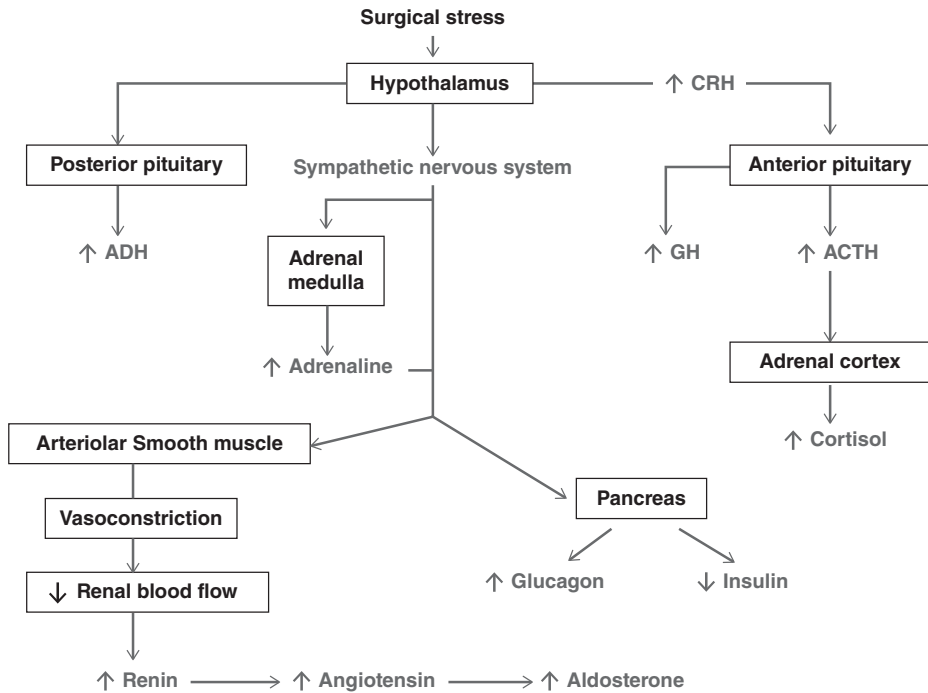


Figure 1.3.1 An overview of the surgical stress response coordinated by the hypothalamus, including the HPA axis, sympathoadrenal and sympathorenal responses. This figure was published in *British Journal of Anaesthesia Education*, 20, Cusack B, Buggy DJ, Anaesthesia, analgesia, and the surgical stress response, 321–328, Copyright Elsevier (2020).

Can you describe the sympathoadrenal response?

Try to answer these questions from first principles.

This is mediated via the hypothalamus and the autonomic nervous system with the release of adrenal medullary catecholamines. There is also increased pre-synaptic nor-adrenaline release. This leads to cardiovascular stimulation with resultant tachycardia and hypertension. Catecholamine effects on other organs include increased brain alertness, increased plasma lactate, increased basal metabolic rate, suppression of insulin release, increased glucagon release and release of renin from the renal juxtaglomerular apparatus. The renin-angiotensin system stimulates aldosterone release, which leads to sodium and water retention.

Can you describe the role of the hypothalamic–pituitary–adrenal axis?

Hypothalamic releasing factors stimulate the anterior pituitary to release adrenocorticotrophic hormone (ACTH), growth hormone and prolactin. Release of ACTH stimulates the adrenal cortex, increasing the production of cortisol. Growth hormone enhances protein synthesis and inhibits protein breakdown, stimulates lipolysis and antagonises insulin. Prolactin is also released, but its role is unclear. Levels of other anterior pituitary hormones including thyroid stimulating hormone remain essentially unchanged.

The posterior pituitary produces increased levels of arginine vasopressin or ADH, which causes water retention, concentrated urine, and potassium loss.

What roles does cortisol play?

Cortisol modulates the overall stress response by its effects on protein catabolism and by its anti-inflammatory effects. Cortisol also antagonises the effects of insulin and growth hormone and has negative feedback effects on the release of CRH from the hypothalamus and ACTH from the anterior pituitary.

Cortisol is released from the adrenal cortex after stimulation by ACTH. This leads to a state of catabolism with protein breakdown, lipolysis, increased glucose production and decreased glucose uptake in peripheral tissues.

Cortisol also exerts anti-inflammatory effects by inhibiting leucocyte migration into damaged areas and inhibits synthesis of various inflammatory mediators including prostaglandins.

Glucocorticoids are required for catecholamines to exert their pressor effects.

What are cytokines, and how do they act?

Cytokines are glycopeptides whose role is in signalling between cells of the immune system and haemopoietic systems. They are produced by activated lymphocytes, macrophages, fibroblasts and endothelial cells in response to tissue injury. Cytokines can be pro-inflammatory (interleukin [IL]-6, and tumour necrosis factor [TNF]- α) or anti-inflammatory (IL-4 and IL-10). Cytokines play an important role in mediating immunity and inflammation by acting on surface receptors of target cells. The effects of the cytokine activation are mostly local but can be systemic. Effects include the production of the acute phase response, fever, granulocytosis, haemostasis, tissue damage limitation and promotion of healing.

What is the acute phase response comprised of?

The acute phase response is the body's response to cytokines produced by the stress response, in particular IL-6, IL-1 and TNF. It comprises increased hepatic synthesis of acute phase proteins such as C-reactive protein, D-dimer, ferritin and complement components. Hepatic synthesis of other proteins such as albumin and transferrin is reduced. Raised prostaglandin E (PGE) levels lead to fever. The response also leads to increased vascular permeability, leucocyte activation and further activation of the hypothalamic-pituitary axis.

What are the potential detrimental effects that the stress response may have?

- Protein catabolism may lead to dramatic muscle loss.
- Hypertension and tachycardia can stress the myocardium and produce ischaemia in susceptible individuals.
- Hyperglycaemia can lead to an impaired immune response and poor tissue healing. It can cause an osmotic diuresis and can lead to organ dysfunction.
- Electrolyte disturbances, particularly hypokalaemia, can cause muscle weakness and promote arrhythmias.

- Fluid overload as a result of increased ADH activity and exogenous fluid administration can cause congestive cardiac failure. Fluid overload can also impair wound healing and can lead to anastomotic breakdown and ileus.
- The pro-coagulant state can increase the likelihood of DVT or PE and can make coronary or cerebral ischaemia more likely.
- Altered gastrointestinal motility can cause nausea, vomiting, diarrhoea or constipation and in the medium term can lead to problems with nutrition.
- Immunosuppression increases the likelihood of poor wound healing and increases susceptibility to nosocomial infection.

What is the significance of the stress response for anaesthetists?

The stress response occurs in the majority of patients that anaesthetists deal with in theatre. In elective cases, the onset of the stress response is predictable. In emergency cases, the stress response may well be under way at the time of anaesthetic induction. The goal for the anaesthetist is to try and minimise the undesirable consequences of the stress response.

How can the stress response be modulated?

The stress response can be influenced by choice of anaesthetic technique and anaesthetic agents. It can be modulated by choice of surgical technique. Other methods that may have a role are nutrition, hormone therapy and temperature control.

Tell me how anaesthesia may influence the stress response.

Think of the drugs you use for anaesthesia and what effects they may have.

The use of various anaesthetic agents may influence the stress response.

Propofol

Propofol suppresses cortisol secretion, likely at the level of the adrenal glands. A single induction dose suppresses circulating cortisol. A continuous infusion may completely inhibit cortisol secretion.

Etomidate

Etomidate inhibits adrenal 11- β -hydroxylase and, therefore, reduces cortisol production. This occurs for up to eight hours following a single induction dose. Use of etomidate is contentious, as it has been shown to increase mortality when used as an infusion in intensive care patients.

Volatile agents

Volatile anaesthetic agents inhibit ACTH, cortisol, catecholamine and GH. They have a number of immunosuppressive and immunomodulating effects, including decreased NK cell activity, decreased cytokine release, decreased neutrophil cell numbers.

Benzodiazepines

Benzodiazepines may have an inhibitory effect on steroid production at the hypothalamic–pituitary level, but the significance of this is not known.

Alpha-2 adrenoreceptor agonists

Clonidine and dexmedetomidine may decrease sympathoadrenal and cardiovascular responses to surgery.

Opioids

High-dose opioids can be used to attenuate the stress response.

Injected opioids have been shown to block ACTH release as a result of reduced CRH release at the hypothalamic level. Alfentanil and fentanyl have been shown to reduce cytokine levels after abdominal surgery. However, the relatively large doses required lead to profound respiratory depression postoperatively and may increase the need for postoperative respiratory support. An immunomodulatory effect has been described with morphine, fentanyl, remifentanyl, methadone and codeine; whereas oxycodone, tramadol, hydrocodone and buprenorphine do not have an immunomodulatory role.

Regional anaesthesia

Using regional anaesthesia may influence the stress response.

Neuraxial blockade is known to inhibit the HPA axis response, by blocking both the afferent activation of the hypothalamus and blocking efferent stimulation of the liver, adrenals and pancreas. Cytokine responses are unaltered, however, as these are stimulated by local tissue damage.

What other methods of altering the stress response are you aware of?

Steroids

Increased levels of endogenous cortisol present in response to surgery can significantly down-regulate IL-6 production. This cytokine response can be suppressed by administering high dose steroids preoperatively. However, there may be an associated increased risk of complications such as hyperglycaemia and wound infection.

Surgical techniques

Using less-invasive surgical techniques (such as laparoscopic or robotic surgery) may reduce the inflammatory response. The duration of surgery, size of incision, and extent of intraoperative manipulation are proportional to tissue injury and the associated stress response. Cytokine release is lower in minimally invasive surgery compared to open techniques.

Nutrition

Enteral feeding has been shown to improve recovery in critically ill patients. Immunonutrition such as the supplementation of feeds with glutamine, arginine and omega 3 fatty acids, may reduce immune-mediated changes seen after surgery. Enhanced recovery after surgery protocols have focussed on minimising fasting times, introducing a carbohydrate load preoperatively and early postoperative enteral feeding.

Temperature control

The metabolic response to hyperthermia is prevented by maintaining a normal temperature throughout the perioperative period.

Further Reading

Burton D, Nicholson G, Hall G. Endocrine and metabolic response to surgery. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2004; 4(5): 144–147.

Cusack B, Buggy DJ. Anaesthesia, analgesia, and the surgical stress response. *British Journal of Anaesthesia Education*. 2020; 20(9): 321–328.

1.3.2 Endocrine Disease and Perioperative Steroid Management – Niladri Das

What are the clinical features of a pituitary tumour?

Pituitary tumours comprise of approximately 10–15% of all intracranial tumours and typically present in one of three ways: hormone hypersecretion syndromes, hormone hyposecretion or mass effect, although the vast majority are incidental radiological findings. The clinical manifestation can often be attributed to tumour size and cell type. Macroadenomas are greater than 10 mm in diameter and may present with features of mass effect to include headache, visual disturbances and vomiting. Larger tumours especially can cause hypopituitarism, cranial nerve palsies and hydrocephalus. Microadenomas are less than 10 mm in diameter and may present with features of hormone excess as in the case of Cushing's disease.

What is acromegaly?

Acromegaly is a chronic, progressive, multisystem disease caused by an excess production of growth hormone by a pituitary macroadenoma. Patients often present with features of local mass effect and hypersecretion of growth hormone. Physical changes likely result from the overgrowth of soft tissue structures to include frontal bossing, prominence of the supra-orbital ridge and jaw, coarse skin, enlarged hands and feet.

What potential issues does acromegaly present for the anaesthetist?

Acromegaly can pose a challenge for the anaesthetist both in terms of the effects of the disease on airway management and body systems.

Meticulous airway assessment in the preoperative period is essential. Airway problems can arise due to soft tissue hypertrophy of the upper respiratory tract, macroglossia and macrognathia. Although airway maintenance is usually straightforward, direct laryngoscopy and tracheal intubation may prove more difficult. In any case, use of an oropharyngeal airway may be helpful during bag-mask ventilation whilst external laryngeal pressure and use of a gum elastic bougie may facilitate tracheal intubation. Videolaryngoscopy and fiberoptic intubation techniques are increasingly being regarded as first line options for anticipated difficult intubation in this patient cohort.

The systemic effects of acromegaly are predominantly respiratory, cardiac and endocrine in nature. Approximately 70% of patients will have obstructive sleep apnoea (OSA) and respiratory function may be further compromised by kyphoscoliosis and proximal myopathy. Acromegalic patients may have refractory hypertension associated with ischaemic heart disease, arrhythmias, cardiomyopathy and congestive heart failure. A preoperative cardiac assessment in the form of a full history, examination and

electrocardiogram should be carried out. Patients with evidence of ischaemic heart disease or left ventricular failure should be further investigated with an echocardiogram. Cardiac function should be optimised prior to an elective surgical procedure. Endocrine features are those of glucose intolerance, diabetes mellitus and thyroid dysfunction. These patients will require management of their blood glucose in the perioperative period. Furthermore, excess peripheral soft tissue deposition may increase the risk of peripheral nerve injury under general anaesthesia. Meticulous attention to patient positioning and padding of pressure areas in theatre is essential. Consideration to post-operative management in a high dependency area should be given, particularly for high-risk cases.

What are the key clinical features of Cushing's disease and its implications for the anaesthetist?

Cushing's disease is caused by hypersecretion of adrenocorticotrophic hormone (ACTH) from a pituitary adenoma leading to an excess of glucocorticoids. It is important to differentiate between Cushing's disease and Cushing's syndrome, the latter referring to a state of chronic glucocorticoid excess regardless of cause. Other causes of glucocorticoid excess may include long-term steroid therapy and adrenal tumours.

Physical manifestations of Cushing's syndrome include truncal obesity, moon facies and thin extremities. Atrophic skin and thin peripheral veins make venous cannulation difficult with a high risk of extravasation. Careful patient handling and positioning is essential in patients with osteoporotic joints.

Cushing's disease predominantly involves the respiratory, cardiac and endocrine systems. With a high incidence of obesity and OSA in this group, patients may have a difficult airway, and respiratory function can be further compromised postoperatively due to proximal muscle wasting. Long-standing refractory hypertension can result in left ventricular hypertrophy and diastolic dysfunction leading to significant haemodynamic instability under general anaesthesia. There is a high risk of perioperative venous thromboembolic disease. Glucose intolerance is seen in nearly two-thirds of patients with Cushing's disease, half of whom will have diabetes mellitus.

What are the important anaesthetic considerations for pituitary surgery?

All patients undergoing transphenoidal pituitary surgery require a thorough preoperative assessment and endocrinology review to elicit the degree of pituitary dysfunction. Importantly, any neurological deficits prior to surgery should be documented. The patient's cardiorespiratory and endocrine function should be fully optimised including the control of blood pressure, blood glucose and hormone therapy as indicated.

The principles of anaesthesia for pituitary surgery are common to those for any neurosurgical procedure. These are to maintain haemodynamic stability, ensure adequate cerebral perfusion and oxygenation in addition to an anaesthetic technique that enables rapid, smooth emergence to allow for early neurological assessment.

The choice of anaesthetic technique is dependent on the surgical approach, patient's comorbidities and past anaesthetic history. The majority of pituitary surgery is now performed via the transphenoidal approach with transcranial resection reserved for large tumours and where the former strategy has failed. Deliberate fracture of the nasal septum

is required for transphenoidal access to the pituitary gland. Nasal bleeding can be minimised through use of a mucosal vasoconstrictor agent. In addition to placing a reinforced tracheal tube, a throat pack can be inserted to prevent contamination of the lower airway with blood and secretions. Antibiotics and intravenous hydrocortisone should be administered following induction of anaesthesia. Balanced anaesthesia can be achieved with either an inhalational or intravenous technique. In addition to standard monitoring, invasive arterial pressure monitoring should be utilised for all cases. The operation can involve periods of intense surgical stimulation where use of short-acting agents, such as remifentanyl, can provide analgesia, intraoperative stability and facilitate rapid recovery from anaesthesia. Pain management is multimodal, although the use of non-steroidal anti-inflammatory drugs remain controversial due to concerns over postoperative bleeding. Postoperative nausea and vomiting are commonplace following neurosurgical procedures and routine pharmacological prophylaxis is recommended.

Complications of transphenoidal surgery, although rare, include persistent leak of cerebrospinal fluid, meningitis, panhypopituitarism, diabetes insipidus, cerebral ischaemia and stroke. A theoretical risk of venous air embolism lies intraoperatively despite the semi-supine operating position and carotid artery injury is rare but potentially devastating. Postoperative airway complications are unusual but patients with a history of OSA are at higher risk and should be monitored in a high dependency environment. It is important to note that continuous positive airway pressure is contraindicated following the procedure due to the risk of tension pneumocephalus. Postoperative neuroendocrine abnormalities may occur temporarily. Hormonal therapy and steroid replacement will be required in all patients after the operation and hence follow-up by an endocrinologist is advised.

You are asked to assess a 45-year-old woman for a total abdominal hysterectomy. She has a diagnosis of Addison's disease. What do you understand about this condition?

Addison's disease is a clinical condition resulting from the destruction of the adrenal cortex. This leads to a deficiency of the adrenal steroid hormones. The majority of cases seen in the UK are due to an autoimmune process. There is an association between Addison's disease and other autoimmune conditions such as pernicious anaemia, vitiligo and thyroid disorders. Alternative causes include adrenal gland malignancy, infection, infarction and haemorrhage into the adrenal tissue.

The clinical features of chronic cases are usually nonspecific and include weakness, fatigue, weight loss and postural hypotension. There may be increased pigmentation of exposed areas and the buccal mucosa. Acute presentations may be witnessed during which the patient is profoundly unwell with abdominal pain, vomiting, dehydration and hypotension. Characteristic biochemical abnormalities are seen; namely hyperkalaemia, hyponatraemia, hypercalcaemia and hypoglycaemia.

How is Addison's disease treated?

Patients require replacement of the adrenal steroids and mineralocorticoids. This is given orally in the form of hydrocortisone and fludrocortisone. All patients should be counselled regarding the importance of taking their medication and carry a steroid alert card. Acute

addisonian crisis is managed with an airway, breathing and circulation approach. Admission to a critical care area should be considered for ventilatory support, intravenous steroid supplementation, fluid resuscitation and correction of serum electrolytes and glucose.

What are the principles of perioperative steroid management in adult patients with adrenal insufficiency?

Patients on long-term replacement corticosteroid therapy, that is daily doses of prednisolone of 5 mg or greater for at least one month in adults, for either primary or secondary adrenal insufficiency are at high risk of adrenal crisis. These patients will require stress doses of hydrocortisone in the perioperative period. Regular medication should be continued up to the time of surgery. An intravenous dose of hydrocortisone 100 mg should be administered at induction of anaesthesia, followed by immediate initiation of a continuous infusion of hydrocortisone at 200 mg/24 hours (alternatively, hydrocortisone 50 mg at 6-hourly intervals) until the patient can resume double their usual dose of oral glucocorticoid. This should then be tapered back to normal dosing within 48 hours in most cases or up to a week for major surgery. It is important to note that dexamethasone is not adequate as replacement therapy in patients with primary adrenal insufficiency as it has no mineralocorticoid activity. Postoperative electrolyte and blood glucose monitoring is imperative.

Further Reading

Menon R, Murphy PG, Lindley AM.
Anaesthesia and pituitary disease,
*Continuing Education in Anaesthesia
Critical Care and Pain*. August 2011; 11(4):
133–137.

Woodcock T, Barker P, Daniel S et al.
Management of glucocorticoids during the
peri-operative period for patients with
adrenal insufficiency. *Association of
Anaesthetists*. February 2020.

1.3.3 Obesity – Caroline SG Janes

Obesity is a growing problem in the United Kingdom and worldwide with a prevalence of over 20% amongst adults. It presents unique challenges to anaesthetists, which you will be expected to be familiar with for the final exam. It is a topic well suited to both the SAQ and SOE and comes up regularly. The podcast on obstructive sleep apnoea complements this topic. In addition, the AoA/SOBA guidelines on the perioperative management of the morbidly obese patient published in June 2015 should be read prior to the exam.

You are asked to see a 38-year-old woman having an umbilical hernia repair pre-operatively. She is 150 cm tall and weighs 120 kg. She has no other comorbidities, takes no regular medication, and has no allergies.

What system is used to classify a person's weight?

The body mass index (BMI) is used as a tool to quantify whether a person is a healthy weight for their height. The equation used is weight in kilograms divided by height in metres squared. WHO uses the following classifications:

- <18.5 is underweight
- 18.5–24.99 is considered normal

- 25–29.99 is overweight
- 30–34.99 is obese 1
- 35–39.99 is obese 2
- 40.0 is obese 3 (which was previously termed as morbid obesity)

The AoA guidelines recommend all surgical patients should have their height and weight measured and recorded and the BMI calculated preoperatively.

Which category of BMI would this patient fall into?

In the long case you will be given all the clinical information beforehand so make sure you work out the BMI if you are given the patient's height and weight.

This woman has a BMI of 53 and is therefore in the obese 3 category. Despite having no reported comorbidities, at this level of obesity it is a multi-system disorder, and she will be a high-risk patient to anaesthetise.

What do you know about fat distribution and the relevance of this to morbidity risk?

Obesity can be of a gynecoid or android fat distribution. Gynecoid fat distribution is more common in women and involves peripheral fat distribution or a 'pear-like' appearance. An android distribution tends to predominate in men, involves a more central or 'apple-like' fat deposition and is associated with a higher morbidity.

Android fat distribution results in fat deposition intraperitoneally and around the neck and airway. Intra-abdominal fat is more metabolically active and is known to be a contributor to several disease states. Hence, patients with this type of visceral obesity are at greater perioperative risk and present greater challenges both to the surgeon and the anaesthetist. These patients are also likely to have metabolic syndrome, which consists of hypertension, insulin resistance and hypercholesterolemia.

Tell me more about the effects of obesity on the respiratory system.

Obesity causes a tendency to hypoxaemia at rest, which is worsened by the supine position and anaesthesia due to the reduction of the functional residual capacity (FRC). FRC declines progressively with increasing BMI, and a patient with a BMI of over 40 will most likely have an FRC of less than 1 litre. Absolute oxygen consumption is increased and obese patients tend to desaturate more rapidly when apnoeic than non-obese patients. In addition, there is decreased chest wall and lung compliance. The closing volume therefore encroaches on FRC leading to airway closure and ventilation–perfusion mismatch. This in turn causes intrapulmonary shunting and hypoxaemia. Laparoscopic surgery and Trendelenburg positioning further aggravate these altered mechanics of breathing in the obese patient. Obese patients therefore require a higher fraction of inspired oxygen and higher ventilatory pressures to prevent desaturation. Positive end-expiratory pressure is especially useful to increase FRC and prevent airway collapse.

Added to this, obese patients are at increased risk of sleep disordered breathing. This describes a spectrum of conditions including obstructive sleep apnoea (OSA) and Obesity Hyperventilation Syndrome (OHS). These are characterised by episodes of apnoea during sleep, snoring and day-time somnolence and they vary in severity. Severe OSA occurs in 10–20% of patients with a BMI >35. These patients also have

increased pharyngeal wall compliance resulting in a tendency of the latter to collapse. OSA ultimately leads to desensitisation of the respiratory centres to hypercapnia and thus increases the sensitivity to opioid-induced respiratory depression. A diagnosis of OSA is associated with a greater than doubling of the incidence of postoperative desaturation, respiratory failure, postoperative cardiac events and ICU admission. It therefore needs to be identified preoperatively and treated appropriately with continuous positive airway pressure (CPAP).

Tell me more about the cardiovascular problems encountered in obese patients.

To meet the increased metabolic needs of being obese, circulating volume, plasma volume and cardiac output are all increased. This increased circulating volume can in turn cause an increase in pre-load that can eventually lead to dilatation and hypertrophy of one or both ventricles. These changes can progress to systemic and pulmonary hypertension, which contribute to the development of coronary heart disease and biventricular failure.

The incidence of arrhythmia, particularly atrial fibrillation, is increased; this is mainly due to sinoatrial node dysfunction and fatty infiltration of the conducting system. Care needs to be taken with ondansetron as the risk of prolonged QT interval is also increased with increasing obesity.

The obese population also carry a higher risk of postoperative deep vein thrombosis. In obese women their risk is 10 times higher than in the non-obese counterparts.

What are the effects of obesity on the metabolic and gastrointestinal systems?

Obesity is a chronic metabolic disorder characterised predominantly by insulin resistance. This is associated with diabetes mellitus and hyperlipidaemia. Obese patients can also develop a fatty liver, sometimes termed steatohepatitis. This is a reversible condition with weight loss but if untreated can progress to cirrhosis and liver failure. Obese patients also have a higher risk of gastric aspiration due to gastro-oesophageal reflux disease and an increased incidence of hiatus hernia.

How would you assess this patient preoperatively?

I would want to conduct a thorough preoperative assessment for this patient. In addition to a routine anaesthetic history, I would specifically look for symptoms or signs suggestive of gastro-oesophageal reflux, cardiovascular or respiratory dysfunction as these can be masked by a sedentary lifestyle. I would use the STOPBANG calculator to ascertain the likely presence of OSA. In my local hospital the pre-assessment criteria are that if the score is ≥ 5 we would then use the Epworth Sleepiness score (ESS). If the ESS score is ≥ 12 then this triggers a referral for sleep studies.

I would perform a thorough assessment of her airway, her back to see whether regional anaesthesia would be feasible and assess veins for venous access.

I would look for previous anaesthetic charts to check for evidence of any airway problems.

A risk prediction calculator should be used for all high-risk patients and the results should be discussed with the patient – I often use the NSQIP ACS risk calculator so this

would be my preference for this patient. For bariatric surgery patients the Obesity Surgery Mortality Risk Score (OS-MRS) calculator can be used.

What investigations would you want for this patient?

I would want a full blood count, urea, creatinine and electrolytes, a fasting glucose level, liver function tests and C-reactive protein and HbA1C if diabetes is known or suspected to be present. I would review her electrocardiogram looking for evidence of ischaemic heart disease or cor pulmonale. If significant cardiovascular disease was suspected, I would request an echocardiogram and a cardiology review. I would also consider an arterial blood gas analysis, lung function tests and, as previously outlined, sleep studies.

What are the practical considerations when listing an obese patient for surgery?

According to the Association of Anaesthetists, all hospitals should have a named consultant anaesthetist and theatre nurse responsible for the management of obese patients. These members of staff are responsible for training all staff to care for these patients and for ensuring availability of specialised equipment. All hospitals should also have local guidelines for the management of the obese patient.

The patient's BMI should be included on the theatre list so that all staff are forewarned. The operating table and ward bed should be checked to ensure that the patient's weight is below the maximum allowable. Additional personnel should be present in theatre to help with positioning and to help turn the patient in an emergency.

Manual handling should be kept to a minimum; therefore the patient should be anaesthetised in theatre on the operating table and on a transfer sheet which can be used with air transfer technology. There should be plenty of gel padding and some wide strapping available. Arm boards should be available for optimum positioning, especially if an extra wide table is not available.

An Oxford Head Elevating Laryngoscopy Pillow (HELP) should be available to aid patient positioning. This helps to ramp the patient so that their tragus is level with their sternum – this reduces the risk of difficult laryngoscopy and improves oxygenation. Difficult airway equipment and videolaryngoscopy should be available and ready to use. A step for the anaesthetist can also be useful for intubation.

Alternative equipment and monitoring devices should be available. An arterial line may need to be inserted in case the non-invasive blood pressure cuff does not read accurately. Intravenous access can sometimes be difficult, therefore an ultrasound machine and central venous access equipment should be immediately available.

As both anaesthesia and surgery are more challenging in obese patients the additional time required should be allowed for when booking the case.

How would you anaesthetise this patient for her umbilical hernia repair?

A patient with a BMI of over 50 should always be anaesthetised by an experienced anaesthetist; I would therefore discuss this case with the duty consultant following my preoperative assessment and confirm the availability of a high dependency bed for postoperative care.

I would administer antacid prophylaxis preoperatively as she will be at increased risk of gastro-oesophageal reflux.

I would make use of the (Society of Bariatric Anaesthesia) SOBA calculator to aid with appropriate drug dosing. My anaesthetic technique would depend on the size of the umbilical hernia. If the hernia is small, I would discuss the possibility of carrying out the surgery under local anaesthesia with the surgical team. If the hernia is large and local anaesthesia is not possible, I would opt for a general anaesthetic technique. I would consider using an epidural for postoperative pain relief although this may be technically difficult. This could provide optimal analgesia, reducing the need for systemic opiates.

I would thoroughly pre-oxygenate the patient, monitoring nitrogen washout and I would induce the patient in a ramped position with abducted arms. If the patient had symptoms of gastro-oesophageal reflux I would use a modified rapid sequence induction with propofol, remifentanyl and rocuronium and secure the airway with a tracheal tube. I would have equipment available in case it is a difficult intubation.

Following intubation I would use pressure-controlled ventilation with volume of 6 ml/kg of lean body weight, and apply sufficient positive end-expiratory pressure. I would fully monitor the patient in accordance with the AoA guidelines maintaining balanced anaesthesia with TIVA with propofol and remifentanyl. I would use boluses of rocuronium guided by the use of a continuous neuromuscular monitor.

If epidural anaesthesia was not possible, I would ask the surgeon to infiltrate the wound with local anaesthetic at the end of the procedure. Ultrasound guided rectus sheath blocks and transversus abdominis plane blocks could also be considered in this event. I would also administer intravenous paracetamol, diclofenac, longer-acting opiates as appropriate and fluid, ensuring the patient is well hydrated.

I would ensure correctly sized intermittent calf compression devices are applied intraoperatively to the patient and discuss starting prophylactic low molecular weight heparin immediately postoperatively with the surgeon.

I would extubate the patient awake and sitting up following reversal of neuromuscular blockade with sugammadex. The patient may benefit from postoperative continuous positive airway pressure (CPAP) therapy. Further analgesia on the high dependency area could be provided with patient-controlled analgesia using fentanyl or morphine but should be carefully monitored.

As the patient is of childbearing age, I would enquire whether she is taking the oral contraceptive pill and advise her that this would have been rendered ineffective by the anaesthetic. If a sugammadex guidance leaflet was available, I would ensure she was given one of these in recovery for later reference.

How is drug-dosing affected by obesity?

As most of the excess weight in obese patients is made up of fat which has a relatively low blood flow, drug-dosing in obesity needs to be adjusted based on the pharmacological properties of the drug. Drugs can be administered according to four types of weight categories: Total body weight (TBW), Ideal body weight (IBW), Lean body weight (LBW) or Adjusted body weight (ABW).

TBW is the actual weight of the patient and is used for drugs such as suxamethonium and low molecular weight heparins (LMWH). IBW is calculated using the Broca

formula – Men (height in cm – 100) and women (height in cm – 105), IBW is not often used to guide drug dosing in anaesthesia. Lean body weight exceeds IBW and plateaus at around 100 kg for a man and 70 kg for a woman. Drugs which should be administered according to LBW include propofol induction dose, fentanyl and alfentanil, morphine, NMBDs, paracetamol and local anaesthetics. ABW is calculated by adding 40% of the excess weight to the IBW and is used for propofol and remifentanyl infusions, sugammadex and most antibiotics. The SOBA calculator is a great resource to aid in drug dosing in obese patients with a BMI > 35.

What special considerations should there be for obese patients postoperatively?

Depending on the extent of the hernia, the length of surgery and the patient's conduct during surgery, it may be necessary for them to be nursed on high dependency or intensive care postoperatively. It will be necessary for there to be a multidisciplinary approach to their care with the involvement of physiotherapists, dieticians, and occupational therapists if they undergo major surgery.

Initially day-case surgery was thought to be unsuitable for obese patients, but, with careful patient selection it is becoming more common and can be very successful. The decision should be made on a case-by-case basis and depends on other comorbidities and the type of surgery.

Respiratory problems may persist into the postoperative period. Supplemental oxygen, breathing exercises, physiotherapy and occasionally non-invasive ventilation may be required to alleviate atelectasis and retained secretions and thus prevent the development of a postoperative chest infection. If a patient with sleep apnoea uses a CPAP machine at home they should be asked to bring this in and should be used in the immediate postoperative period.

Analgesia should be multimodal including paracetamol and NSAIDs should be used routinely if there are no contraindications. Regional or peripheral nerve blockade should be used if possible and appropriate. Intramuscular injections are best avoided in obese patients as the drug uptake is unpredictable. Opiates can be used with care although they are best avoided in patients known to have OSA.

Many obese patients are diabetic and will require blood glucose monitoring perioperatively. In major surgery a sliding scale with insulin and dextrose to maintain normoglycaemia may be required until the patient resumes a normal diet. This may also be necessary in some non-diabetic patients during the catabolic phase post-surgery.

Obese patients should be nursed postoperatively on a special bariatric bed with a pressure-relieving mattress. Early mobilisation is essential as is postoperative DVT prophylaxis. Well-fitting thromboembolic deterrent (TED) stockings and subcutaneous LMWH should be used routinely even in minor surgery. In major surgery continuation of DVT prophylaxis post-discharge is now mostly routine.

Further Reading

Peri-operative Management of the Obese
Surgical Patient 2015. *The Association of*

Anaesthetist of GB and Northern Ireland.
Society for Obesity and Bariatric
Anaesthesia. March 2015.

1.3.4 Perioperative Management of Diabetes – Andrea Binks

A 45-year-old obese woman is scheduled for knee replacement surgery. She has a history of non-insulin dependent diabetes and is treated with oral hypoglycaemic agents.

What would be your anaesthetic management for this patient?

Try and classify this answer. Dividing into preop, periop and post op works well here.

I will discuss this patient's management by thinking about preop assessment, perioperative management and postoperative instructions.

I would start with preoperative assessment.

In addition to my standard perioperative assessment there are two further areas I would concentrate on. The first is glycaemic control and the second is assessment of diabetic complications.

Starting with glycaemic control, I would ask about the patient's recent diabetic control, at home and in hospital and reviewing any relevant charts at the patient's bedside. I would also make an assessment of hydration and acid base balance by clinical examination, review of fluid balance charts and by checking an arterial blood gas if appropriate.

Current hypoglycaemic agents should be reviewed, including all oral hypoglycaemic agents and any additional insulin regime. In the acute setting, poorly controlled sugars can cause symptomatic hypoglycaemic episodes or severe dehydration with an acidosis caused by either lactate or by ketoacids.

High blood sugars perioperatively are linked with an increase in wound infection rate and, in cardiac surgery, with poor neurological outcome.

An HbA1c should be checked; ideally the patient should have levels of less than 69 mmol/L (8.5%) before referral for elective surgery. A referral to a diabetes specialist would be appropriate if HbA1c is greater than 69 mmol/L or 8.5%. HbA1c should be optimised in the three months prior to elective surgery. In patients with poor diabetic control, the surgery should be postponed unless it is immediately lifesaving.

At the preop visit, a management plan should be made in conjunction with the patient and endocrine team if appropriate. Written instructions should be given to the patient as to how to manage their diabetic medication. See Tables 1.3.4.1 and 1.3.4.2.

In the long term, poor glycaemic control is associated with more microvascular complications of the disease.

What diabetic complications would you look for?

Try and discuss these in the order of relevance.

Firstly I would assess the patient for coronary artery disease as it is 4–5 times more common in patients with diabetes and may be asymptomatic, or present with atypical symptoms.

The second complication I would assess is autonomic neuropathy.

There are two major manifestations of this, which are of particular concern to the anaesthetist.

The first is disordered cardiovascular responses and orthostatic hypotension. They may have unexpected tachyarrhythmias or hypotension unresponsive to ephedrine.

Table 1.3.4.1 Perioperative management of oral hypoglycaemic agents

Drug class	Mechanism of action	Day prior to admission	Patient for AM surgery	Patient for PM surgery	When to restart
Fasting – require <i>omission</i> due to risk of hypoglycaemia					
Sulphonylureas (e.g. glibenclamide, gliclazide)	Bind to the SUR 1 subunit of the K ⁺ /ATP channel in the pancreatic β cell membrane and increase insulin secretion	Take as normal	Omit morning dose if fasting	Omit on day of surgery	Restart when eating and drinking
Metaglinides (e.g. repaglinide or nateglinide)		Take as normal	Omit morning dose if fasting	Give morning dose if eating am	Restart when eating and drinking
Fasting – require <i>omission</i> due to risk of ketoacidosis					
SGLT2 inhibitors (e.g. dapagliflozin, empagliflozin)	Block glucose reabsorption in the proximal convoluted tubule of the kidney increasing glycosuria	Omit	Omit	Omit	Restart if well and eating normally
Fasting – may require <i>adjustment</i>					
Biguanides (e.g. metformin)	Improve insulin sensitivity and reduce hepatic gluconeogenesis. Also enhance GLP-1 secretion and modulate bile acid recirculation	Take as normal	If taken once or twice a day – take as normal. If taken three times per day – omit lunchtime dose		Restart when eating and drinking
Alpha glucosidase inhibitors (eg acarbose)	Slow intestinal carbohydrate absorption by inhibiting pancreatic α amylase and intestinal α glucoside hydrolase enzymes in small intestine	Take as normal	Omit morning dose if fasting	Give morning dose if eating am	Restart when eating and drinking
Fasting – can be <i>continued</i>					
Thiazolenediones (e.g. pioglitazone)	Activate PPAR gamma receptors to increase insulin sensitivity in fat and muscle	Take as normal	Take as normal	Take as normal	Restart when eating and drinking

Table 1.3.4.1 (cont.)

Drug class	Mechanism of action	Day prior to admission	Patient for AM surgery	Patient for PM surgery	When to restart
GLP1 agonists (e.g. exenatide, liraglutide, daily/ weekly admin)	Increase glucose dependent insulin secretion, reduce glucagon secretion, inhibit stomach emptying	Take as normal	Take as normal	Take as normal	Take as normal
DPP IV inhibitors (e.g. sitagliptin, vildagliptin, linagliptin)	Inhibit DPP IV enzyme from inactivating incretins GIP and GLP-1	Take as normal	Take as normal	Take as normal	Restart when eating and drinking

If eGFR<60 or procedure requires contrast media – omit day prior to surgery and for 48 hours post op

Hypotension tends to be poorly tolerated in these patients, particularly if there is underlying cardiovascular or cerebrovascular pathology.

Gastroparesis is the second concern. Despite fasting, gastric volumes can be quite large, thus increasing the risk of reflux and aspiration on induction.

How would you assess for the presence of autonomic neuropathy?

I would assess for autonomic neuropathy by asking the patient to perform a Valsalva manoeuvre. In the presence of autonomic neuropathy, the blood pressure will drop and because the ability to compensate is lost, the blood pressure will stay low until the intrathoracic pressure is released.

What other diabetic complications would you ask about?

The third complication I would ask about is, diabetic nephropathy. If there is any evidence of this, for example microalbuminuria, then the risk of postop renal failure is greatly increased. I would ensure that appropriate haemodynamic monitoring was in place and that fluid balance is maintained. Diabetic nephropathy can also be associated with anaemia and platelet dysfunction.

Next, I would assess for peripheral neuropathy as this affects approximately 20% of diabetic patients, and is characterised by pain, paraesthesia and sensory loss. It is important to document the level of neurological deficit prior to surgery, particularly where regional or neuraxial anaesthetic techniques are being considered.

I would then ask about stiff joints, as 30–40% of patients will have some limited joint mobility. This may lead to difficulties with laryngoscopy and tracheal intubation.

I would ask the patients about eye complications, as patients with retinopathy are at high risk of developing vitreous haemorrhage during hypertensive episodes.

I would then ascertain if there were any associated medical comorbidities such as obesity, with the increased risk of obstructive sleep apnoea and fatty liver disease, or other autoimmune disease such as hypoadrenalism, myasthenia gravis or thyroid disease.

Table 1.3.4.2 Perioperative management of insulin therapy

LONG-ACTING INSULIN				
Insulin	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
Once daily long acting (morning)	Abasaglar [®] Humulin I [®] Insulatard [®] Insuman Basal [®] Lantus	No dose adjustment needed	Give 80% of morning dose and check blood glucose on admission	Give 80% of morning dose and check blood glucose on admission
Once daily long acting (lunchtime)	Levimir [®] Semglee [®] Tresiba [®] Toujeo [®] Xultophy [®]	Give 80% of lunchtime dose	Restart insulin at normal dose when eating and drinking	Restart insulin at normal dose when eating and drinking
Once daily long acting (evening)		Give 80% of evening dose	No dose adjustment necessary	No dose adjustment necessary
Twice daily long acting		Morning dose as normal, Give 80% of evening dose	Give 80% of morning dose and check blood glucose on admission Evening dose unchanged	Give 80% of morning dose and check blood glucose on admission Evening dose unchanged
PREMIX INSULIN PREPARED BY MANUFACTURERS				
Insulin	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
Twice daily (premixed insulin)	Humulin M3 [®] Humalog Mix 25 [®] Humalog Mix 50 [®] Hypurin Porcine (30/70 Mix [®]) Insuman Comb 15 [®] Insuman Comb 25 [®] Insuman Comb 50 [®] Novomix 30 [®]	No dose adjustment necessary	Half usual morning dose. Blood glucose to be checked on admission Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of usual mixed insulin	Half usual morning dose. Blood glucose to be checked on admission Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of usual mixed insulin

Table 1.3.4.2 (cont.)

LONG-ACTING INSULIN				
Insulin	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
Three times daily (premixed insulin)		No dose adjustment necessary	Half usual morning dose. Blood glucose to be checked on admission Omit lunchtime dose Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of usual mixed insulin	Half usual morning dose. Blood glucose to be checked on admission Omit lunchtime dose Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of usual mixed insulin
SELF MIXED INSULIN PREPARED BY PATIENT/CARER				
INSULINS	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
Twice daily (two different types of insulin combined by the patient/carer into one injection)	Short-acting: Actrapid® Apidra® Fiasp® Humalog® Humulin S® Hypurin® Porcine Neutral Insulman Rapid® Lyumjev® Novorapid® AND Intermediate acting: Humulin I® Hypurin® Porcine	No dose adjustment necessary	Calculate the total dose of both morning insulins and give half of this as intermediate acting insulin only, in the morning Blood glucose to be checked on admission Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only	Calculate the total dose of both morning insulins and give half of this as intermediate acting insulin only, in the morning Blood glucose to be checked on admission Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose

Table 1.3.4.2 (cont.)

LONG-ACTING INSULIN				
Insulin	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
	Isophane Insulatard [®]		component of usual mixed insulin	If not eating, give basal only component of usual mixed insulin
SHORT-ACTING INSULIN				
Insulins	Example medications	Day prior to admission	Patient for AM surgery	Patient for PM surgery
Short-acting insulin with meals (two to four doses a day)	Actrapid [®] Apidra [®] Fiasp [®] Humalog [®] Humulin S [®] Hypurin [®] Porcine Neutral Insulman Rapid [®] Lyumjev [®] Novorapid [®]	No dose adjustment necessary	Omit morning dose if no breakfast is eaten Blood glucose to be checked on admission Omit lunchtime dose if not eating and drinking normally Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of insulin	Take usual morning insulin dose with breakfast Omit lunchtime dose if not eating. Blood glucose to be checked on admission Resume normal insulin with evening meal if eating normally If eating a half/small meal, give half usual dose If not eating, give basal only component of insulin
Resume taking usual insulin the morning after surgery. However, blood glucose levels may be higher than usual for a day or so.				
Variable rate intravenous insulin infusions	Dose of long-acting insulin should be 80% Short-acting, intermediate and premixed insulins should be discontinued and replaced by a long-acting basal insulin at a dose of 0.2 units per kilogram A return to the patients' normal diabetes management can be made when they are eating and drinking normally. Adjustments may need to be made to insulin doses as insulin requirements may change in the postoperative period. Blood glucose levels should be monitored and advice sought from the specialist diabetes team if necessary			

Table 1.3.4.3 Management of intraoperative hypo or hyperglycaemia

Intraoperative hyperglycaemia (CBG >12 mmol/L)	Intraoperative hypoglycaemia (CBG: 4.0–6.0 mmol/L)
Check ketones	
<i>Type 1 diabetes</i>	CBG 4.0–6.0
Subcutaneous rapid-acting insulin given (up to 6iu*)	50 ml 20% glucose (10g) given iv
<i>Type 2 diabetes</i>	
Subcutaneous rapid-acting insulin 0.1iu/kg (up to max 6 iu*)	CBG <4.0
*Assume a 3 mmol/L drop in CBG per 1 iu insulin	100 ml 20% glucose (20g) given iv

How would you optimise this woman’s glucose control intraoperatively?

I would ensure that ideally, she be listed first on the surgical list, or at least in the first third of the list. This is in order to minimise fasting time. Prolonged fasting will lead to catabolism and higher risk of needing a variable rate insulin infusion (VRII) with its associated risks.

In the perioperative period, blood sugars should be checked regularly. For this patient having elective surgery, blood sugars should be measured on admission, at induction of anaesthesia and at least hourly throughout the surgery if on insulin or insulin secretagogues, otherwise minimum 2-hourly. Capillary blood glucose (CBG) levels in the range of 6–12 mmol/L are acceptable if the patient is managed with diet alone, or in the range of 4–12 mmol/L if the patient is managed with oral hypoglycaemic agents that don’t cause hypoglycaemia.

How would you manage a blood sugar level outside this range?

If the CBG is less than 6, intervention with oral or intravenous carbohydrate should be considered. If the CBG is more than 13 on two or more readings, and the patient is usually on SGLT2 inhibitors, then blood ketone levels should be checked. If there is no DKA, then a dose of subcutaneous rapid-acting insulin can be administered (Table 1.3.4.3)

A variable rate IV insulin infusion should be started if fasting results in more than one missed meal.

Which patients will benefit from a variable rate IV insulin infusion (VRII)?

Patients with poorly controlled diabetes, patients who will miss more than one meal, and patients with Type 1 diabetes will need to have a VRII commenced. It is recommended that patients with Type 1 diabetes continue their long-acting insulin at 80% of their usual dose whilst on a VRII. (Further information in Table 1.3.4.2). All other antidiabetic agents should be withheld with the exception of GLP1 agonists which can be continued as normal.

How would you manage a patient with a continuous subcutaneous insulin infusion (CSII)?

Patients with a CSII should be encouraged to maintain their usual background rate and to self-correct to a CGM of 6–10 mmol/L. If the patient is unable to do this, or is likely to miss more than one meal, or to be too unwell to manage their CSII in the perioperative period, then the CSII should be discontinued and a VRII should be commenced instead.

For a patient to continue their CSII throughout the perioperative period, they should ensure that the infusion catheter is plastic (not metal), that the infusion site is distant from the operative field and from the diathermy site and is accessible at all times by the anaesthetist. This will require planning preoperatively as the infusion site is changed every 2–3 days, and acquisition of the appropriate catheter may take some time.

What principles will be guiding your choice of anaesthetic technique?

My aim for this woman is to have her eating and drinking normally as soon as possible after her surgery so she can restart her oral hypoglycaemic medications. In order to facilitate this, I would strongly consider a neuraxial or regional anaesthetic. If this is contraindicated, I would make sure I am using short-acting anaesthetic agents, multimodal analgesia and antiemetic prophylaxis.

What are the benefits and risks of neuraxial or regional techniques for this patient?

The benefits of a regional or neuraxial technique are reducing venous thromboembolism risk, reducing bleeding and a reduction in the surgical stress response. It also facilitates an early return to normal diet, especially when used as the sole technique. Regional anaesthesia can also be used as part of a multimodal analgesia plan.

The potential risks are an increase in haemodynamic instability, especially in patients with autonomic neuropathy, an increased risk of infective complications, and an increase in peripheral neuropathy when using a regional nerve block.

How would you approach her fluid management intraoperatively?

My goals for fluid management intraoperatively are to maintain intravascular volume, ensure renal perfusion and to maintain normal electrolyte balance. There is no evidence suggesting any balanced crystalloid solution is better than any other; however, Hartmann's solution has been shown to be safe without contributing to any clinically significant hyperglycaemia.

If a variable-rate insulin infusion is needed, then a balanced crystalloid solution should be given to maintain electrolyte balance. The patient may well need a glucose-containing solution in addition in order to prevent proteolysis, lipogenesis and ketogenesis.

What instructions would you give to the recovery staff regarding glucose control?

I would ask the recovery staff to continue checking her CBG hourly. I would ask that they hand over to the ward staff that once she is eating and drinking normally, she can restart her oral hypoglycaemic medications. (see Table 1.3.4.2)

How would you define diabetic ketoacidosis?

Diabetic ketoacidosis is a state of insulin insufficiency that results in high blood sugar levels and the accumulation of organic acids in the blood. It is a serious, often life-threatening complication of diabetes and is characterised by the triad of hyperglycaemia, metabolic acidosis and ketonaemia.

What is the mechanism for ketone production in diabetes?

This is testing knowledge of some of the steps in intermediary metabolism. You need to at least be able to explain the final steps, which lead to metabolic acidosis.

Ketones are produced from acetyl CoA in the liver mitochondria. Acetyl CoA is the end product of β -oxidation of fatty acids, and the product of condensation of two pyruvate molecules. Acetyl CoA can be combined with oxaloacetate and then enter into the citric acid cycle of aerobic metabolism. In states of excess fatty acid breakdown such as diabetic ketoacidosis and starvation, then there is not enough oxaloacetate to combine with all the Acetyl CoA molecules. The excess acetyl CoA molecules are diverted into ketone production. The accumulation of the ketoacids betahydroxybutyrate and acetoacetate are what result in the development of a metabolic acidosis.

Can you explain the pathophysiology of sodium-glucose cotransporter-2 (SGLT2) associated ketoacidosis?

SGLT2 receptors are expressed on the surface of pancreatic α -cells and are responsible for detecting glucose, and the regulation of glucagon secretion. Inhibiting SGLT2 receptors in the proximal tubules of the kidney promotes glycosuria and therefore reduces plasma glucose. This leads to a reduction in insulin secretion and further increases plasma glucagon levels. The shift in the insulin: glucagon ratio stimulates lipolysis, hepatic fatty acid oxidation and increased ketogenesis in the liver.

In the context of starvation and surgical stress, the additional presence of adrenaline and cortisol result in insulin resistance, protein catabolism, further lipolysis and ketogenesis. The ongoing renal glucose excretion maintains euglycemia.

Further Reading

Association of Anaesthetists of Great Britain and Ireland. Peri-operative management of the surgical patient with diabetes 2015. *Anaesthesia* 2015; 70: 1427–1440.

Centre for Perioperative Care. Guideline for perioperative care for people with diabetes mellitus undergoing elective and emergency surgery. CPOC, 2021. https://cpoc.org.uk/sites/cpoc/files/documents/2021-03/CPOC-Diabetes-Guideline2021_0.pdf [Accessed 29 April 2022].

McGinlay M, Mruthunjaya S. Anaesthetic management of diabetes. *Anaesthesia and Intensive Care Medicine*. 2017; 18: 481–487.

Partridge H, Perkins B, Mathieu S, et al. Clinical recommendations in the management of the patient with type 1 diabetes on insulin pump therapy in the perioperative period: A primer for the anaesthetist *British Journal of Anaesthesia*. 2016; 116: 18–26.

Robinson C, McGinlay M, Mruthunjaya S. Perioperative management of diabetes. *Anaesthesia and Intensive Care Medicine*. 2020; 21: 548–557.

Thiruvankatarajan V, Meyer EJ, Nanjappa N et al. Perioperative diabetic ketoacidosis associated with sodium-glucose co-transporter-2 inhibitors: A systematic review. *British Journal of Anaesthesia*. 2019; 123: 27–36.

1.3.5 Calcium and Magnesium Homeostasis – Farzad Saadat and Sarah F Bell

Magnesium and calcium may be discussed together due to the physiological association. Magnesium is used in a number of different contexts by the anaesthetist, requiring good knowledge of its applications, risks and physiology. Calcium is a core physiology question, which you should remember from primary FRCA.

You are working on the delivery suite, and are asked to review a 31-year-old patient, 36 weeks gestation, who is in labour with confirmed pre-eclampsia. She is already taking IV labetalol, but is complaining of headaches, and is found to have brisk reflexes.

What is the next course of treatment for this patient?

This could be an opening question leading to speaking about magnesium, a common topic in the FRCA.

This patient is displaying neurological features of pre-eclampsia. As well as good blood pressure control, appropriate treatment is IV magnesium. I would give the patient a loading dose of magnesium of 4 g over 15 minutes followed by 1 g per hour as an infusion.

Any patient on a magnesium infusion requires regular monitoring for signs of magnesium toxicity; which include loss of deep tendon reflexes, weakness and shortness of breath. Measurement of plasma magnesium levels should be considered with a therapeutic range of about 2–3 mmol/L.

The patient's symptoms should be monitored for any deterioration as well as blood tests to screen for HELLP syndrome or renal impairment. Other supportive therapy needs to be considered, senior obstetricians and anaesthetists notified and consideration of expedited delivery of the baby.

Tell me more about hypermagnesemia.

The effect depends on the plasma level. At plasma concentrations of 4–5 mmol/L the patient may lose their tendon reflexes and suffer from muscle weakness. At levels of 7–8 mmol/L the patient will have respiratory muscle paralysis and at levels over 10 mmol/L cardiac arrest will occur.

Treatment is aimed at reducing levels. Calcium gluconate will antagonise the effects of the magnesium whilst diuresis and dialysis will act to remove the excess.

Can you tell me where most magnesium is located in the body and what the normal plasma concentration is?

It is predominantly an intracellular ion with an intracellular concentration of 5–20 mmol/L. The normal plasma concentration ranges from 0.5 to 1 mmol/L.

What are the roles of magnesium?

Magnesium is an ion that has a number of therapeutic applications. You need to have detailed, accurate knowledge of its actions.

Magnesium is important in maintaining electrical potentials across cell membranes, it is vital in the function of ATP and the synthesis of DNA, RNA and proteins. It affects calcium metabolism, and hypomagnesaemia is often associated with hypocalcaemia. Intracellular magnesium also inhibits calcium influx and it is therefore described as a physiological calcium antagonist.

Magnesium affects most of the body systems. It is a cardiovascular depressant and can cause a reduction in cardiac output and vascular tone. It has antiarrhythmic effects and inhibits the release of catecholamines. Magnesium is an anticonvulsant that reduces excitability of nerves and antagonises calcium at the presynaptic junction. Magnesium will cause skeletal muscle weakness and in theory precipitate respiratory failure at high enough plasma levels. It is an effective bronchodilator. With regards to the genitourinary system, magnesium is a tocolytic and a mild diuretic. Finally, magnesium reduces platelet activity.

How is magnesium stored and how are levels controlled?

Magnesium is the second most common intracellular cation, after potassium. It is distributed 65% in bone and 35% in cells. About one third of dietary magnesium is absorbed in the small intestine. The kidneys control the plasma levels by controlling excretion of magnesium. Magnesium is reabsorbed in the ascending limb of the loop of Henle, with only 1% excreted in the urine. Parathyroid hormone enhances both gut and kidney reabsorption, whilst aldosterone increases renal excretion.

What are the causes of abnormal magnesium levels?

Magnesium deficiency is due to magnesium loss from diarrhoea or due to lack of intake, classically in patients receiving TPN. Raised magnesium levels may be caused by excessive treatment or intake.

What are the effects of hypomagnesemia?

Paraesthesia, fits, tetany and arrhythmias have all been observed. It is important to remember that hypocalcaemia may also occur. The treatment is replacement of magnesium.

What are the therapeutic actions of magnesium?

Magnesium has a number of therapeutic roles. It is used in acute asthma to treat bronchospasm. The British Thoracic Society recommends 2 g IV magnesium sulphate given over 20 minutes for adults with acute severe asthma who have not had a good response to initial bronchodilator therapy.

Magnesium is used as an antiarrhythmic in torsades de pointes and ventricular arrhythmias unresponsive to other treatment. Again, a 2 g IV bolus should be given but over 10 minutes.

Magnesium is also used during surgery for removal of pheochromocytoma in order to suppress catecholamine release.

As well as pre-eclampsia magnesium is also indicated for eclamptic seizures and 4 g is again given as a bolus, although this is reduced if the woman is already on an infusion.

Magnesium can be used as an adjunct to analgesia, as it may have an effect on the NMDA receptor.

Finally, magnesium is used in cases of tetanus to reduce spasm and autonomic instability. It is again given as a bolus followed by an infusion titrated to symptoms and plasma levels.

Are there any anaesthetic considerations when administering magnesium?

As a calcium antagonist, magnesium can induce or worsen existing hypotension, and should be used cautiously in cases with cardiovascular instability. In addition, it can prolong the length of neuromuscular blockade, as calcium antagonism can affect the pre-synaptic release of acetylcholine.

Magnesium is classified as a calcium antagonist, what are the actions of calcium?

Discussion of magnesium could lead to further questions about calcium. Questions regarding the management of hyper or hypocalcaemia can occur on their own.

Calcium is vital for a number of different body systems. In the haematological system calcium is required for haemostasis. In the musculoskeletal system it is vital for the structural integrity of bone. It plays a key role in the release of acetylcholine at the presynaptic bulb and is also integral to the function of the actin-myosin power-stroke in skeletal muscle contraction. Calcium is also important in cardiac and smooth muscle contraction. Finally in the neurological system, calcium functions as a neurotransmitter and as a second messenger system.

How is calcium metabolism controlled?

Calcium metabolism is controlled by parathyroid hormone, vitamin D and calcitonin.

Parathyroid hormone is secreted in response to low plasma calcium levels. It causes a rise in plasma calcium and a fall in plasma phosphate. The parathyroid hormone increases bone resorption, enhances vitamin D activity and also reduces phosphate resorption by the kidney.

Vitamin D is produced in the skin in response to sunlight. It is activated in the liver and then kidney to form 1,25 dihydroxy vitamin D. This active form of vitamin D is stimulated by low calcium and phosphate levels and by parathyroid hormone. Active vitamin D increases calcium and phosphate levels by increasing gut absorption and bone resorption. The active vitamin D has a negative feedback effect on parathyroid hormone levels. Calcitonin reduces plasma calcium and phosphate levels.

What are normal plasma calcium levels?

The normal level is 2.2–2.6 mmol/L. It is important to remember that calcium is 40% bound to albumin and it is the unbound, ionised portion which is active. Calcium

measurements should therefore be adjusted for the albumin level. For every 4 g/L of albumin that is below the normal level of 40 g/L, 0.1 mmol/L of calcium should be added to the initial result.

What are the causes of hypocalcaemia?

Calcium levels need to be considered in conjunction with phosphate levels. If calcium and phosphate are low the cause may be removal of the parathyroid tissue, chronic renal failure, hypoparathyroidism or pseudohypoparathyroidism (failure of the target cell response to parathyroid hormone). If the phosphate level is normal or raised the cause might be osteomalacia, overhydration or pancreatitis.

What are the features of hypocalcaemia?

Patients may present with tetany, perioral tingling, carpo-pedal spasm and depression. Neuromuscular excitability may also be seen and can be elicited by tapping over the facial nerve causing twitching – this is Chvostek's sign. The ECG findings would be of an increased QT interval.

How can we treat hypocalcaemia?

Mild hypocalcaemia can be treated with oral calcium. Severe symptomatic hypocalcaemia should be treated with intravenous calcium gluconate 10%, repeated as necessary.

What are the possible causes of hypercalcaemia?

The most common causes of hypercalcaemia are malignancy and primary hyperparathyroidism.

What are the features of hypercalcaemia?

The mnemonic 'bones, stones, abdominal groans and psychic moans' helps to remind you of the different systems!

Abdominal symptoms of pain, vomiting, weight loss, constipation are all features. As is polyuria, polydipsia, renal failure and renal stones. Patients may also develop depression and confusion. Furthermore, hypertension and eventually cardiac arrest may ensue. The ECG may reveal a reduced QT interval.

How would you treat hypercalcaemia?

Calcium levels greater than 3.5 mmol/L or patients with symptomatic hypercalcaemia require treating. Treatment includes rehydration, diuretics and bisphosphonates.

1.3.6 Hypokalaemia and Hyperkalaemia – Farzad Saadat and Sarah F Bell

You are most likely to discuss plasma electrolyte disturbances as part of a blood result in a long case or when looking at an ECG. You might also be asked to discuss your initial management of an abnormal electrolyte result.

Whilst attending pre-op assessment clinic, you are called by one of the clinic nurses, who wishes to discuss a patient's blood tests. The 45-year-old patient is due for an elective knee arthroscopy next week, and his routine blood results have shown a serum potassium concentration of 3.0 mmol/L. The patient reports having been unwell for the last few days. What are the possible causes of hypokalaemia? How would you start to ascertain the cause in this patient?

Potassium balance is a core topic that you should remember from the primary FRCA. Try and structure your answer whenever possible.

Hypokalaemia may be due to inter-compartmental shifts, increased potassium loss or reduced intake. Inter-compartmental shifts may be due to alkalosis, insulin, beta-2 agonists and hypothermia. Increased losses may be caused by diuretics, mineralocorticoids, renal tubular acidosis, ketoacidosis, ileal conduit, diarrhoea and vomiting, sweating and dialysis. Reduced intake may be due to lack of potassium in IV fluids or diet.

I would take a full medical history, paying particular attention to the recent illness and signs of potassium loss such as vomiting or diarrhoea. I would explore their drug history, looking for drugs that can cause compartmental shifts of potassium such as insulin or anti-psychotic drugs. Finally, I would look for causes of increased urinary losses, and look at his renal function, and look for drug causes such as diuretics.

Would you proceed with surgery in this case?

Patients with hypokalaemia are at risk of developing arrhythmias. Elective operations should be cancelled if the potassium level is below 3.0 mmol/L. Given the non-urgent nature of this patient's surgery, it could be delayed until the cause is identified and corrected. If the patient's recent history of illness were found to be the most likely cause, it is likely the potassium losses are already correcting. I would ask the patient to represent for blood tests, and if we found the potassium levels to be self-correcting, I would proceed with surgery.

How would you manage a hypokalaemic patient presenting for urgent, emergency surgery?

Emergency procedures may warrant more rapid correction with ECG monitoring in a high dependency environment. The maximum rate of potassium administration via central venous access is 40 mmol per hour. Plasma levels would need to be closely monitored and I would aim to achieve a level of 4.0 to 5.0 mmol/L. This is particularly important for patients taking digoxin due to an increased risk of toxicity if the level is below 4 mmol/L.

Let's talk about physiology. How is potassium regulated in the body?

Potassium intake is approximately 50 to 150 mmol per day. Potassium is predominantly an intracellular ion, present in concentrations 20 to 30 times higher than in plasma. The plasma potassium concentration is 3.5 to 4.5 mmol/L.

Acute regulation of plasma potassium levels is achieved by the actions of insulin which promotes uptake of potassium into cells. Chronic regulation of plasma potassium levels is achieved by the kidney. Normally all of the filtered potassium is reabsorbed in the proximal tubule. Active secretion occurs in the distal tubule and collecting duct. Aldosterone acts on the sodium potassium ATPase pump in the distal convoluted tubule to increase potassium excretion. Its release is stimulated by hyperkalaemia and via the renin-angiotensin pathway.

What are the ECG changes of hypokalaemia?

This question may be posed in the context of an abnormal ECG.

The ECG changes include T wave flattening and inversion, U waves, ST depression a prolonged PR interval and increased QT interval. There may be progression to arrhythmias and possible asystole.

What are the effects of hypokalaemia?

A reduction in myocardial contractility can lead to heart failure. Generalised skeletal muscle weakness, respiratory muscle weakness or muscle cramps may be a feature, along with ileus and polyuria.

How can we treat hypokalaemia?

Treatment is aimed at replacement of potassium which can be oral or intravenous depending on the severity of the condition, as well as treatment of any complications and identification and correction of any underlying cause.

IV potassium replacement can be given peripherally, but high concentrations should be given centrally. In addition, central administration of potassium requires ECG monitoring.

What are the causes of hyperkalaemia?

This is a common, significant condition that you will doubtless have encountered and treated. Detailed knowledge will be expected. Make sure you structure your answer to show your knowledge!

They can be split into inter-compartmental shifts, reduced excretion and increased intake. Causes of inter-compartmental shifts include acidosis, rhabdomyolysis, trauma, malignant hyperthermia and suxamethonium use in patients with burns and spinal injury. Reduced excretion may be due to renal failure, adrenocortical insufficiency and drugs such as ACE inhibitors or potassium-sparing diuretics. Increased intake might be due to excessive IV potassium or as a consequence of a massive blood transfusion.

What are the ECG changes of hyperkalaemia?

The ECG changes include peaked T waves, wide QRS and prolonged PR interval which may progress to loss of P waves, ST depression, ventricular fibrillation and asystole. These changes are potentiated by hypocalcaemia, hyponatraemia and acidosis.

What are the effects of hyperkalaemia?

The patient may present with muscle weakness or gastrointestinal symptoms of nausea, vomiting and diarrhoea. Furthermore, they may present with palpitations or cardiovascular collapse.

How can we treat hyperkalaemia?

A potassium level of 6.5 mmol/L or more or ECG changes consistent with hyperkalaemia require urgent treatment to avoid deterioration and cardiac arrest. Treatment is aimed at cardiac protection, shifting potassium into the cells, stopping further potassium administration and removal of excess potassium. Calcium chloride 10 ml of 10% intravenous will provide some cardiac protection by acting as a physiological antagonist to the potassium. 10 IU insulin in 50 ml 50% dextrose infusion given over 30 minutes, or a similar regimen, will drive the potassium into the cells and therefore reduce the plasma potassium level. Additional emergency treatment might include beta two agonists such as salbutamol, which will also move potassium into the intracellular space and dialysis to remove potassium from the plasma. Less acute treatment of hyperkalaemia may include calcium resonium, orally or rectally.

What are the anaesthetic implications of hyperkalaemia?

Hyperkalaemia will predispose the patient to arrhythmias which may be fatal intraoperatively. It is therefore essential to treat symptomatic hyperkalaemia prior to induction of an anaesthetic, as I described earlier. Considerations during anaesthesia would then involve avoidance of suxamethonium (which would transiently increase the potassium level by 0.5 to 1 mmol/L and might be fatal). Acidosis and hypothermia would also worsen the hyperkalaemia by encouraging the shift of potassium from the intracellular space into the plasma. Controlled ventilation would allow potential correction of pH via optimisation of carbon dioxide levels. An arterial line would allow regular monitoring of electrolyte and acid base status. Neuromuscular blockade may be prolonged and therefore should be monitored.

1.3.7 Hyponatraemia and Hypernatraemia – Farzad Saadat and Sarah F Bell

Disorders of sodium balance are core topics and can be asked about in a variety of contexts; ICU, findings prior to day surgery or in emergency cases or TURP syndrome.

You are the SPR on the intensive care unit. The nursing staff ask you to review a 58-year-old man who was admitted 2 days ago following a large intracranial bleed. He had a craniotomy on admission but has shown little signs of recovery since. The nursing staff are concerned as his latest blood tests show a sodium concentration of 152 mmol/L.

What are the possible causes of hypernatraemia in this case?

Hypernatraemia is defined as a plasma sodium level of greater than 145 mmol/L. There are several potential causes in this case, but among my differentials here would be

iatrogenic (through administration of high sodium infusions such as hypertonic saline), dehydration due to inadequate fluid administration, water depletion greater than sodium (which may be caused by renal diuresis from osmotic diuretics such as mannitol, glucose or urea) or neurogenic diabetes insipidus (DI).

How would you begin your review, given his serum sodium concentration?

I would perform a full assessment of the patient and pay particular attention to his fluid balance. I would assess his fluid status by looking at his mucus membranes, his capillary refill time and any invasive cardiac monitoring he might have. I would want to look at his fluid chart, in particular to look for his urine output to check for diuresis, and to assess which fluids he has been given since admission. I would want to establish the timing of the rise in his serum sodium levels, to establish how quickly it has risen, if it coincided with any given treatment or was pre-existing.

Are there any investigations you would order to investigate his hypernatraemia?

In order to investigate DI, I could order a urine specific gravity test, a repeat serum sodium and paired serum and urine osmolalities; a high serum osmolality, greater than 305 mmol/kg and low urine osmolality, less than 350 mmol/kg are suggestive of DI.

What can you tell me about diabetes insipidus?

Diabetes insipidus is due to impaired water resorption from the kidney. It may be cranial or nephrogenic in origin. Cranial DI is caused by reduced ADH secretion from the posterior pituitary and nephrogenic DI is due to impaired response of the kidney to ADH. Cranial DI may develop after a head injury or infection, pituitary tumour or autoimmune disease. Nephrogenic DI may be due to infection of the kidney, hypokalaemia or drugs such as lithium or democlocycline.

Investigations to confirm DI should reveal hypernatraemia with high plasma osmolality and low urine osmolality. Outside of ICU a water deprivation test should confirm the diagnosis. A normal response to water deprivation would be an increase in urine osmolality but a patient with DI would have an abnormally dilute urine. In cranial DI, the administration of desmopressin (synthetic ADH) would cause an increase in urine osmolality whereas with a nephrogenic DI no such response should occur. The treatment would depend on finding the cause of the DI and attempting to correct this.

Your examination finds the patient to be polyuric with a urine output of over 300 ml/hour and a diagnosis of DI is confirmed by your investigations. How would you treat this case?

There are two aims of treatment: to replace lost fluids and to replace ADH. I would ask the nurses to monitor his urine output and replace his hourly losses with NG water.

If significant polyuria continues, I would consider administration of desmopressin. Continuous monitoring of serum sodium levels is important as rapid correction of hyponatraemia can lead to pulmonary or cerebral oedema.

What other changes in sodium regulation might you observe after a head injury?

This question has the potential to get very confusing. Try and stick to basic physiology.

Both SIADH and cerebral salt wasting syndrome might occur. In both of these diseases the sodium level may fall. In SIADH the excess ADH would cause increased water reabsorption and a dilutional hyponatraemia with increased total body water. In cerebral salt wasting syndrome excessive sodium and water loss occur together.

Moving back to hypernatraemia, and thinking about it in other contexts, what are its effects?

The symptoms of hypernatraemia depend on the rate of increase and the plasma level reached. The patient may present with the signs and symptoms of dehydration (including thirst, nausea, tachycardia, hypotension, confusion and lethargy). Central nervous system effects may also include seizures, muscle spasms, hyperreflexia and possibly intracranial haemorrhage. The patient may be hyperthermic. Pre-renal failure can occur due to a fall in cardiac output.

How should hypernatraemia be treated?

The treatment of hypernatraemia depends on the cause. If water excess is suspected then diuretics would be appropriate. If water depletion is evident then rehydration with fluids containing minimal sodium would be required. If sodium depletion is the cause, then treatment with 0.9% saline is necessary. Patients suffering from diabetes insipidus might need desmopressin.

What are the anaesthetic implications of hypernatraemia?

Elective surgery should be postponed if the sodium level is above 155 mmol/L due to the potential effects of sodium changes in intracerebral fluid compartments. If the patient requires emergency surgery, then a central line should be placed and cautious correction of the sodium level with regular checks should be commenced.

Now let's move onto low sodium levels. What are the causes of hyponatraemia?

Hyponatraemia is defined as a plasma sodium level below 135 mmol/L. This may be due to water excess, sodium redistribution, water excess disproportionate to sodium excess or water depletion with greater sodium deficiency.

Water excess may be further split into causes of excess administration of hypotonic IV fluids, drinking too much water or TUR syndrome; or reduced water excretion in the case of SIADH, drugs (such as oxytocin) or the normal physiological response to surgery.

Unbalanced sodium and water excess may be evident in patients with nephrotic syndrome, heart failure or hepatic failure.

Unbalanced sodium and water deficiency is predominantly due to renal compromise due to diuretics, renal tubular acidosis or hypoadrenalism; but it may also be due to diarrhoea and vomiting or pancreatitis. Finally, redistribution of sodium into the intracellular space may be found in patients with hyperglycaemia.

What are the effects of hyponatraemia?

The speed of onset will have some impact on the severity of the condition. Plasma sodium levels of 125–130 mmol/L tend to present with gastrointestinal upset whilst levels below 125 mmol/L lead to neurological symptoms of lethargy, headache, seizures, psychosis and coma. Respiratory depression and muscle weakness may also occur.

How should hyponatraemia be treated?

Management is aimed at treating the underlying cause and cautious correction of the hyponatraemia. This may include fluid restriction and diuretics. In cases of SIADH, democycline may offer some relief. The administration of hypertonic saline should be carefully considered due to risk of inadvertent over or rapid correction. Rapid correction of hyponatraemia may lead to central pontine myelinolysis, especially when the hyponatraemia has been chronic.

What are the anaesthetic implications of hyponatraemia?

Severe hyponatraemia (i.e. less than 125 mmol/L) is a contraindication to elective procedures. In emergency cases severe hyponatraemia should be corrected cautiously with ECG monitoring and regular plasma levels to help guide resuscitation. Invasive arterial and central venous pressure monitoring may also be indicated and the patient nursed in a high dependency unit or intensive care.

What can you tell me about transurethral resection of the prostate or TURP syndrome?

This is a surgical complication that you should be aware of. Try and explain the cause of the problem as this will indicate to the examiner that you really understand the condition.

TURP syndrome occurs in about 5% of operations of transurethral resection of the prostate. The resection of the vascular prostate tissue opens venous sinuses which allow absorption of hypotonic glycine 1.5% irrigation fluid into the systemic circulation. The volume of fluid absorbed depends on the duration of the procedure, hydrostatic pressure of the irrigation fluid (or height of the bag above the patient), the venous pressure of the patient, for example, if dehydrated, and vascularity of the prostate.

What are the features of TURP syndrome?

There are three classical features of TURP syndrome: firstly, a dilutional hyponatraemia; secondly fluid overload and thirdly glycine toxicity. The symptoms and signs of TURP syndrome are predominantly cardiovascular and neurological. Cardiovascular effects

include tachycardia and hypertension followed by hypotension, angina and cardiovascular collapse. Neurological features include a burning sensation in the face and hands, confusion, convulsions and coma. A regional anaesthetic technique with an awake patient potentially allows the anaesthetist to monitor the neurological features more closely since they would be masked by general anaesthesia.

How would you treat TURP syndrome?

Treatment involves recognition and resuscitation of the patient. Communication with the surgeon to terminate surgery as soon as possible is essential. Initial management would involve an airway, breathing and circulation approach with assessment and treatments occurring simultaneously. If the patient has airway compromise the airway will need to be secured with an endotracheal tube. Breathing complications such as pulmonary oedema may also necessitate intubation and controlled ventilation. Oxygen should be administered to the awake patient. Treatment of fluid overload and hyponatraemia involves stopping IV fluids and commencing fluid restriction. Furosemide will promote a diuresis and is indicated if pulmonary oedema is present. Antiarrhythmics and vasopressors may be needed to combat arrhythmias and hypotension. Hyponatraemia causing encephalopathy will require cautious correction of plasma sodium levels with fluid restriction and possibly hypertonic saline. N-methyl D-aspartate (NMDA) receptor activity is also potentiated by glycine, which paradoxically may precipitate encephalopathy and seizures. Magnesium (whose plasma level may also be reduced through dilution) exerts a negative control on the NMDA receptor and also has a membrane-stabilising effect. It should therefore be considered as part of the therapy for seizures in TURP syndrome. Invasive patient monitoring should be considered. Due to the potential development of seizures and cerebral oedema, anticonvulsive therapy and measures to reduce raised intracranial pressure may be instituted.

Can you tell me more about hypertonic saline?

Hypertonic saline is indicated when several symptoms develop or serum sodium levels fall below 120 mmol/L. Ideally sodium levels should rise by 1 mmol per hour (and not more than 20 mmol over 48 hours). Once sodium levels reach 125 mmol/L or symptoms cease, hypertonic saline should be stopped. The patient will need to be closely monitored during this treatment. A suggested infusion regimen is 1 ml per kilogram per hour of 3% saline to produce a rise of 1–2 mmol/L per hour in a 70 kg man.

Further Reading

Hirst, C, Allahabadia, A, Cosgrove, J. The adult patient with hyponatraemia, *British Journal of Anaesthesia Education*. 2015; 15 (5): 248–252.

O'Donnell, AM, Foo, ITH. Anaesthesia for transurethral resection of the prostate, *Continuing Education in Anaesthesia Critical Care and Pain*. 2009; 9(3): 92–96.

1.3.8 Hypothermia – Ashley C Davis

You are asked to assess a patient in recovery who has just had a laparotomy. Their core temperature is 33°C.

What is hypothermia?

It is a fall in bodily core temperature to $<35^{\circ}\text{C}$, and it can be classified into mild, moderate or severe. Mild $<33\text{--}35^{\circ}\text{C}$ and severe $<28^{\circ}\text{C}$.

What is the inter-threshold range?

The inter-threshold range is the range of core temperatures over which no autonomic thermoregulatory responses occur. It is normally approximately 0.2°C to 0.4°C .

How does general anaesthesia alter the inter-threshold range?

General anaesthesia widens the inter-threshold range considerably. The thermoregulatory threshold to cold is decreased by approximately 2.5°C and on the upper range the threshold is increased by approximately 1.3°C .

Why are patients under anaesthesia hypothermic?

The normal responses to heat loss are both autonomically and behaviourally mediated. During general anaesthesia the patient will be unable to move to a warmer environment, exercise, shiver or put more clothes on. The hypothermia associated with anaesthesia occurs in a triphasic pattern as depicted in Figure 1.3.8.

Phase 1 – ‘Redistribution’. The initial rapid decline in temperature is due to redistribution. Anaesthetic agents cause vasodilation, which results in a redistribution of warm blood to the peripheries, and cool blood from the peripheries enters the core circulation. Furthermore, with the effect of the widened inter-threshold range, there is delayed activation of compensatory vasoconstriction.

Phase 2 – ‘Heat loss’. This gradual fall in temperature is due to heat loss mechanisms which exceed the gain from metabolic heat production. Anaesthesia reduces the metabolic rate by 15–40%, as muscle activity and brain metabolism are decreased. Heat loss occurs to the environment via physical mechanisms.

Phase 3 – ‘Plateau’. At this stage heat loss is matched by metabolic heat production. This may occur when the patient becomes sufficiently hypothermic to activate compensatory vasoconstriction.

Can you describe how regional anaesthesia affects heat loss?

Regional anaesthesia results in a reduction in shivering and inhibits vasoconstriction below the level of the block. As demonstrated in Figure 1.3.8 the initial hypothermia occurs due to redistribution of warm core blood to the peripheries, secondary to the vasodilation induced by regional anaesthesia. Vasoconstriction in areas not affected by the regional block may compensate partially. The most profound risk of hypothermia occurs during combined neuraxial and general anaesthesia.

What are the physical mechanisms by which heat has been lost from the patient?

This question is testing your knowledge of basic science as well as its clinical application.

Heat loss may occur through radiation, convection, evaporation, conduction, and through respiration.

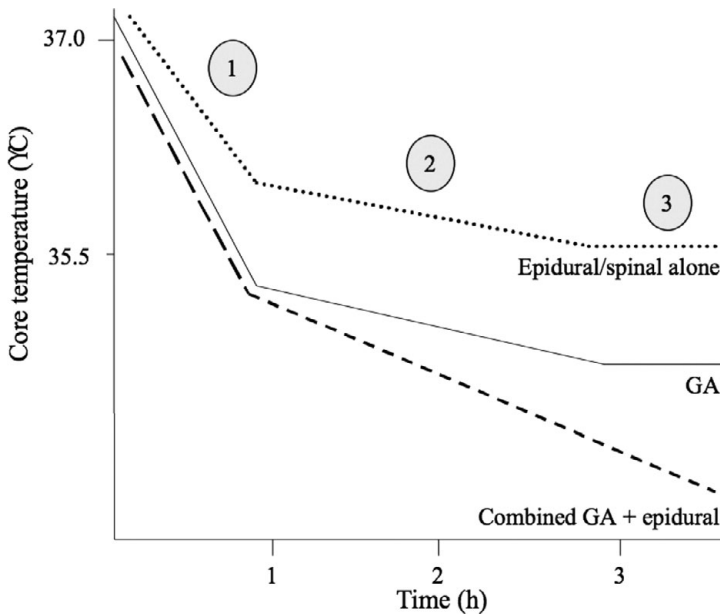


Figure 1.3.8 The triphasic patterns of hypothermia during neuraxial, general, or combined neuraxial and general anaesthesia (GA). Patients under combined neuraxial and GA are higher risk for more profound hypothermia. This figure was published in the British Journal of Anaesthesia, 3, Kirkbride DA, Buggy DJ, Thermoregulation and mild peri-operative hypothermia, 24–28, Copyright Elsevier (2003).

What is radiation?

Radiation is the transference of heat from a hot object to a cooler object. The extent of heat loss depends on the differential between the room temperature and the body temperature. Radiation accounts for 40 to 50% of heat loss during anaesthesia.

What is convection?

The air layer closest to the body is warmed by conduction. As the air temperature increases, the air rises and is carried away by convection currents. This accounts for about 30% of heat loss, but can be accentuated if the body is exposed to convection currents, for example laminar flow systems.

What is evaporation?

As the moisture on the body's surface evaporates, it loses the latent heat of vaporisation, and the body cools down. This accounts for 20–25% of heat loss. The process is accelerated if there is a large moist area exposed during surgery, for example major abdominal or major orthopaedic procedures.

What is conduction?

Conduction is a process by which heat is passed directly from a warm object to a cooler object by direct contact. During anaesthesia this accounts for only 3–5% of heat loss unless the body is touching an efficient heat conductor such as a metal table.

Table 1.3.8 Physiological responses to hypothermia

Body System	Response to Hypothermia
CVS	Initially tachycardia, peripheral vasoconstriction, increased cardiac output. Followed by sinus bradycardia, ECG changes (J-waves), increased risk arrhythmias.
Resp	Initially tachypnoea, followed by reduction in minute volume and in severe cases apnoea. Impaired hypoxic pulmonary vasoconstriction.
CNS	Decreased cerebral blood flow and cerebral metabolic rate of oxygen. Isoelectric EEG at 20°C.
Haem	Oxygen-haemoglobin dissociation curve shifts to left – decreased oxygen delivery. Blood viscosity increased – risk of intravascular stasis. Enzyme systems of the extrinsic and intrinsic coagulation pathways inhibited resulting in coagulopathy.
Metabolic	Decreased metabolic rate of ~6% for every degree fall in core temperature. Enzyme reactions and intermediary metabolism impaired below 34°C may result in prolonged drug action (especially neuromuscular blocking drugs).
Renal	Diuresis due to increased renal blood flow. Failure of renal tubular reabsorption of sodium and water.
GI	Impaired liver function; ileus; pancreatitis. Hyperglycaemia due to reduced insulin secretion and peripheral glucose utilisation.

Can you describe how heat is lost through respiration?

During respiration, heat is lost through evaporation and by warming inspired air. This can account for up to 10% of heat loss. During anaesthesia this process can be minimised by use of heat and moisture exchangers.

What are the physiological and clinical consequences of hypothermia?

Try to be systematic here.

The physiological effects of hypothermia principally affect the cardiorespiratory, haematological, neuromuscular, gastrointestinal, renal, and metabolic systems, as outlined in Table 1.3.8.

The clinical complications of perioperative hypothermia include: shivering in recovery leading to increased oxygen consumption and pain; greater blood loss in theatre and consequent need for blood transfusion; angina, myocardial infarction, ventricular tachycardia and cardiac arrest; increased incidence of wound infection; pressure sores and a prolonged stay in recovery.

How would you manage the patient in recovery?

Hypothermia of gradual onset is best managed with a slow rewarming process at about 1°C per hour. Heat can be applied to the patient using external or internal methods. The

easiest method of rewarming is by using forced warm air heating blankets or radiant heaters. Alternatively the patient can be rewarmed by using warm intravenous, intragastric or intraperitoneal fluids, or by bladder irrigation via a urinary catheter. The most efficient way of rewarming, but by far the most invasive, is to put the patient on cardiopulmonary bypass or another extracorporeal circuit.

Where have you seen hypothermia being used in a theatre setting?

The examiner is looking to see that you have had exposure to a wide range of anaesthetic specialties.

Hypothermia is used in cardiac surgery when the patient is put onto cardiopulmonary bypass. If hypothermia is used, the patient is typically cooled to a temperature of 28–32°C. In more complex cardiac surgery, for example surgery on the aortic root, deep hypothermia is induced where the patient is cooled to a temperature of 15–22°C, which allows periods of low blood flow or circulatory arrest whilst providing cerebral protection. Deep hypothermic circulatory arrest can also be induced for complex vascular, neurosurgical and urological procedures.

Further Reading

Kirkbride DA, Buggy DJ. Thermoregulation and mild peri-operative hypothermia. *British Journal of Anaesthesia*. 2003; 3(1): 24–28.

Kuht J, Farmery AD. Body temperature and its regulation. *Anaesthesia and*

Intensive Care Medicine. 2021; 22(10): 657–662.

Riley C, Andrzejowski J. Inadvertent perioperative hypothermia. *British Journal of Anaesthesia Education*. 2018; 18(8): 227–233.

1.3.9 Hyperthermia – Steffan Morgan and Ami Jones

You are called to A&E to assess a 35-year-old patient who has run a half marathon. He is drowsy and his temperature is 42°C. The A&E registrar is concerned with the hyperthermia and thinks he needs high dependency care.

What is hyperthermia?

Hyperthermia is defined as an acute condition that occurs when the body produces or absorbs more heat than it can dissipate, resulting in a rise in core body temperature.

What can cause hyperthermia?

Be general and brief.

Causes of hyperthermia can be environmental, drug related or related to a medical condition such as sepsis or an acute burn injury. It can also be classified by excessive heat production, reduced heat dissipation and hypothalamic dysfunction.

What form of hyperthermia is this patient suffering from?

This patient is suffering from environmental hyperthermia. This can range from mild heat exhaustion to the more extreme heat stroke. Heat stroke exists in two forms;

classical heat stroke and exertional heat stroke. This patient is likely to be suffering from exertional heat stroke, which typically occurs acutely in young fit and healthy patients who have taken part in exercise during high ambient temperatures. Classical heat stroke occurs over several hours or days and tends to occur in the elderly and infirm when ambient temperatures are raised for several days.

How do you differentiate between heat exhaustion and heat stroke?

Heat stroke requires the presence of hyperpyrexia and neurological dysfunction. It typically involves temperatures of greater than 40°C and the patient is often confused or unconscious and can be hypotensive, suffer from cardiac dysrhythmias and even cardiorespiratory arrest.

If your diagnosis of heat stroke is correct in this patient, how would you manage him?

I would assess his airway, breathing and circulation; make an assessment of his conscious level and commence cooling of the patient. I would start by externally cooling the patient – place him in a cold environment if possible and begin surface cooling him with cold towels, ice packs and I could consider cold water immersion. I would aim to achieve a target temperature of no less than 39°C . Measuring of the core temperature using a rectal or oesophageal probe is often indicated. As he is severely hyperthermic I would consider the use of internal cooling methods such as cold intravenous fluids, cold peritoneal and stomach lavage. Intravascular cooling techniques such as haemofiltration or cardiopulmonary bypass can be considered; however, they are usually not necessary.

Apart from cooling the patient are there any other therapies that may be indicated?

Heat stroke patients are often intravascularly depleted and require fluid resuscitation guided by cardiovascular parameters and urine output. Cooling reduces heat-induced vasodilatation, and over-resuscitating with fluid can cause circulatory overload, cardiac failure and pulmonary oedema, especially if the patient has pre-existing heart disease. The patient may therefore also require inotrope or vasopressor support. Benzodiazepines can also be used to prevent shivering and treat seizures.

What tests might you order on this patient?

The differential causes of hyperthermia are vast and many tests may be required to find an underlying cause. A sepsis screen, bloods including full blood count, U&Es, liver function tests, thyroid function tests, creatine kinase and a coagulation screen should be taken. If appropriate a toxin screen should also be performed.

What is this patient at risk of if their temperature is not reduced in a timely manner?

Temperatures of more than 40°C can be destructive to the brain and ultimately progress to multiorgan failure and death. A response similar to that which occurs in

the systemic inflammatory response syndrome occurs and can affect all organs within the body resulting in seizures, coma, dysrhythmias, hypotension, disseminated intravascular coagulation, rhabdomyolysis, hepatic failure and even cardiorespiratory arrest.

Your CT2 calls you to theatre as they are concerned that the patient on the operating table having an appendicectomy has a very high temperature. The oesophageal probe in situ is recording a temperature of 40°C. What is your differential diagnosis?

As with the other questions, divide these into patient factors, environmental factors etc . . .

Causes of a high temperature intraoperatively include patient-related, drug-related and environmental-related factors.

The patient is undergoing an appendicectomy so has a potential source of sepsis. Intra-abdominal abscesses can cause significant febrile response which often settles once source control is gained. The patient may also be suffering from sepsis from another source such as a pulmonary or urinary tract infection. Pulmonary embolus can also cause a febrile response. Hyperthyroidism, either pre-existing or acute undiagnosed, should also be considered, as should phaeochromocytoma.

Drug-related factors particular to anaesthetic agents include malignant hyperpyrexia associated with the inhalational anaesthetic drugs and suxamethonium. If the patient usually takes anti-depressants or anti-psychotics they are also at risk of serotonin or neuroleptic malignant syndrome. A febrile response can also occur in response to the administration of blood products or drugs such as N-acetylcysteine.

Environmental factors are those such as a very high ambient temperature or humidity, excessive warming by warm intravenous fluids, air warming blankets or warming mattresses.

What further information would you require?

Think history, investigation and examination.

Further information that I would require would include an assessment of the patient's medical notes; from these I would be able to discover if there were any pre-morbid conditions of note or any current medications, transfusions or recreational drugs which may be implicated in the pyrexia. There may also be a family history of malignant hyperpyrexia recorded in the anaesthetic history or history of problems during previous anaesthetics, although this should have been identified when choosing an appropriate mode of anaesthesia and indeed, I would make a note of any drugs given during the anaesthetic which may have triggered a malignant hyperpyrexia.

I would want to determine the time frame within which the pyrexia has arisen. Examining the observations prior to coming to theatre may show a pre-existing pyrexia which may suggest a septic origin. If the pyrexia has developed *de novo*, I would want to ascertain how quickly it has risen intraoperatively. A temperature that has gradually increased over the course of 2 to 3 hours is likely to be of a very different origin than one that has increased by a number of degrees over a shorter time period.

I would review any blood tests that had been recorded prior to theatre as raised white cell count or raised C-reactive protein would also suggest a septic origin.

I would also review the patient's vital signs as a tachycardia and high blood pressure would be more in keeping with a malignant hyperpyrexia rather than a sepsis. Further information may be gained from the patient's end-tidal carbon dioxide and inspired/expired oxygen levels as a patient suffering from malignant hyperpyrexia would be hypermetabolic and have an increased oxygen requirement and carbon dioxide production rate out of keeping with that which would be expected from a high temperature alone. A stepwise increase in CO_2 is classical of malignant hyperthermia. Performing a full clinical examination of the patient may also allow me to elicit signs of sepsis or features consistent with a malignant hyperpyrexia such as muscle rigidity. Blood tests may show an acidosis, hyperkalaemia, raised creatinine kinase and DIC.

You have deduced that the most likely cause of the hyperthermia is malignant hyperpyrexia caused either by the suxamethonium or the volatile anaesthetic agent. Describe your initial management.

This management is based on an AoA document, which should be a well-rehearsed drill.

I would cease all potential precipitant drugs and convert to a malignant hyperthermia 'safe' technique. I would ask the surgeon to stop operating and inform him of my suspicions. I would then call for senior help and reassess the patient's airway, breathing and circulation. I would remove the patient from the anaesthetic breathing circuit and increase minute ventilation to 2–3x normal with 100% oxygen. I would insert activated charcoal filters on both the inspiratory and expiratory limbs of the circle system. I would maintain anaesthesia with an intravenous agent such as propofol for the remainder of the operation. I would ask my anaesthetic assistant to prepare 2–3 mg/kg of dantrolene and have further boluses of 1 mg/kg ready. These can be administered every five minutes. I would commence active cooling of the patient by administering cold intravenous fluids, switching the warming blanket to a cool temperature and ask the surgeon to perform cold peritoneal lavage if the temperature is greater than 39°C . I could also consider extracorporeal heat exchange.

I would insert an arterial line and send blood for gas analysis, potassium, haematocrit, platelet count, clotting and creatine kinase. I would also consider inserting a central venous line and a urinary catheter and perform dipstick urinalysis for myoglobinuria as well as sending a sample to the lab for formal analysis.

How would your management continue for the duration of the operation?

My management from this point would depend on the clinical situation. Surgery should be postponed if possible. Treatment goals are $\text{EtCO}_2 < 6 \text{ kPa}$ with normal minute ventilation and a core temp of $< 38.5^\circ\text{C}$. I would treat hyperkalaemia with insulin/dextrose and calcium chloride. If myoglobinaemia were detected I would consider forced alkaline diuresis aiming for a urine output of more than 2 ml/kg/hr, with a pH of greater than 7.0. If no contraindications are present, then sodium bicarbonate can be given. I would

treat disseminated intravascular coagulation with fresh frozen plasma, platelets and cryoprecipitate as indicated. Cardiac dysrhythmias are usually tachyarrhythmias and can be treated with drugs such as magnesium, amiodarone and shorting acting β -Blockers.

Where would you care for the patient postoperatively?

I would continue invasive monitoring of the patient in the intensive care unit and continue to cool to normothermia and treat evolving symptoms appropriately. Following recovery, the patient will need counselling and referral to the malignant hyperthermia unit in Leeds.

You are called to review a long-term patient on the ICU who is being weaned from a ventilator. He has spiked a temperature of 39.6°C .

What are the physiological effects of pyrexia on this critically ill patient?

Be organised and work through systems methodically.

A fever on the ICU is defined as a temperature of greater than 38.3°C , although a lower threshold should be considered in immunocompromised and neutropenic patients. An increase in body temperature has effects upon each system within the body. It is associated with an increased morbidity and mortality. It increases both oxygen demand and expenditure of energy, approximately 10% for every 1°C increase. This can have a profound effect upon a critically ill patient who may already have insufficient oxygen supplies and will be in a catabolic state and already have high energy demands. The oxyhaemoglobin dissociation curve shifts to the right which improves off-load of oxygen to the tissues. Cardiovascularly the patient is often tachycardic and can be hypotensive which may require fluid resuscitation and inotropic support.

What aspects of the patient's clinical history may be of importance when trying to determine the cause of the fever?

As the patient is a long-term patient any potential infection is likely to be nosocomial in nature. I would want to ascertain how long any invasive lines, catheters or drains have been in situ and determine the duration of ventilation. The route of tracheal intubation is important as a patient who is nasally intubated is at more risk of sinus infections. I would also question the nursing staff regarding the presence of any purulent secretions from tracheal suction or discharge from drain or line sites or indeed any wounds. A history of prior haematological disease, recent foreign travel or prior infection with diseases such as tuberculosis would also be important to ascertain, as would the patient's current acute medical or surgical problems.

What aspects of the patient's clinical examination may give further information?

There may be crepitations or bronchial breathing audible on auscultation of the lungs or dullness to percussion consistent with pleural effusion. There may be a new heart murmur or other evidence of subacute bacterial endocarditis such as splinter

haemorrhages or Janeway lesions. Examination of the skin may show evidence of septic emboli, cellulitis or fungal infection of skin folds. Surgical sites should be inspected for any evidence of infection/collection.

A review of the patient's recent microbiology results may also herald clues as to potential infective pathogens and the trends of inflammatory markers such as C-reactive protein, procalcitonin and platelet count. At present there is insufficient evidence to suggest a procalcitonin level should be used to commence antibiotics. A raised white cell count may also give an indication of an ongoing infective process. High lactate counts are often seen in sepsis. Other tests to consider include serum amylase or lipase in patients with abdominal pain, urine samples and broncho-alveolar lavage.

How might you pharmacologically reduce their temperature?

If infection is present then I would commence antibiotics. Anti-pyretic agents such as paracetamol are often administered with good effect. NSAIDs also have an anti-pyretic effect, and although these drugs are also associated with renal dysfunction, they have been proven to lower body temperature, tachycardia and lactate accumulation in septic patients.

Further Reading

Achaiah NC, Bhutta BS, AK AK. Fever in the Intensive Care Patient. [Updated 2023 Feb 9]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan.

Available from: www.ncbi.nlm.nih.gov/books/NBK570583/.

GuptaPK, HopkinsPM. Diagnosis and management of malignant hyperthermia. *British Journal of Anaesthesia Education*. 2017;17: 249–54.

1.3.10 Thyroid Disease and Thyroid Surgery – Helen L Jewitt and Stephanie Wallis

You are asked to assess a 65-year-old woman for sub-total thyroidectomy. Describe your preoperative assessment.

To answer this question you should use the same approach as for the preoperative assessment of any patient, namely by taking a full history, examining the patient and obtaining appropriate investigations.

Your specific priorities in this case are to elicit evidence of the patient's thyroid status and any evidence of airway compromise as a result of their thyroid disease.

I would begin by taking a history from the patient. In the history I would try to elicit symptoms of hypothyroidism such as lethargy and intolerance to cold, or hyperthyroidism such as agitation, intolerance to heat and weight loss. I would find out the reason for surgery and any medications prescribed for the thyroid problem. It is vitally important to establish any symptoms of airway compromise resulting from the thyroid pathology. This includes asking about shortness of breath, swallowing difficulties and noisy breathing. Exacerbation of symptoms of dyspnoea or stridor on lying flat is an important indicator of a potentially difficult airway.

In addition to these specific points I would take a full anaesthetic, medical and surgical history.

My examination would focus on eliciting signs suggestive of hypo or hyperthyroidism. These may include bradycardia, coarse skin and hair and non-pitting oedema in hypothyroid patients and tachycardia, atrial fibrillation, tremor and eye involvement in patients with hyperthyroidism. I would perform a careful examination of the airway and neck to assess the size of any thyroid swelling, detect obvious deviation of the trachea and establish any retrosternal extension of the mass.

Appropriate investigations in this patient include blood tests; full blood count, urea and electrolytes, thyroid function tests and corrected calcium. A chest X-ray may show deviation of the trachea. This can be further investigated with a CT scan to more accurately locate and estimate the degree of tracheal narrowing. An ECG and echocardiogram may be indicated by features in the history such as palpitations or coexisting cardiac disease. A preoperative nasendoscopy is useful for two reasons. It gives some indication of the view of the larynx likely to be obtained during direct laryngoscopy and allows assessment of vocal cord function prior to surgery.

This woman has a large goitre with evidence of retrosternal extension. She is uncomfortably short of breath on lying flat. She is clinically and biochemically euthyroid. A CT scan of her thorax shows a marked deviation and narrowing of the trachea at the level of the upper sternum. Describe your options for the technique of induction of anaesthesia in this patient.

There are no absolute right and wrong answers to a question like this but your answer should demonstrate that you are aware of the potential problems with the case and have given thought to how to avoid or address them.

This woman has clinical and radiological evidence of airway compromise due to her thyroid swelling. I anticipate her airway may be difficult both in terms of obtaining a view at laryngoscopy and perhaps of greater concern, difficulty passing an endotracheal tube past the narrowing. I would ensure these concerns were fully discussed at the team briefing, outlining a sequential plan for the management of the airway. Airway equipment to facilitate this plan should be prepared and checked, including reinforced endotracheal tubes of a smaller diameter.

There is a lack of consensus about the safest technique to use in this scenario. Options for consideration are an intravenous induction with videolaryngoscopy, inhalational induction or awake fiberoptic intubation. An awake tracheostomy, whilst attractive in terms of controlling the airway, can be potentially hazardous with excessive bleeding from the vascular thyroid tissue.

Having explored the relative pros and cons of each of these options my plan A would be an inhalational induction in a semi-recumbent position using high flow nasal oxygen to optimise preoxygenation. I would use topical local anaesthesia applied to the vocal cords under deep inhalational anaesthesia to facilitate intubation, using a small diameter reinforced tube to negotiate the tracheal narrowing.

A potential airway rescue technique is ventilation via a rigid bronchoscope and in cases with anticipated difficulty I would consider asking for an ENT surgeon to be present during the induction.

Aside from the airway what are the other perioperative concerns with patients having thyroid surgery?

Careful eye protection should be used in patients with eye involvement since it may not be possible to close their eyes fully. There is the potential for significant blood loss and limited access to cannulae in the patient's forearms due to the extended position of the arms under the surgical drapes. An additional large bore cannula with an extension should be available to allow the rapid administration of fluid in the event of haemorrhage.

How would you extubate this patient?

I would extubate the patient sitting up, spontaneously breathing and fully awake, having ensured adequate reversal of neuromuscular blockade. I would consider the possibility of airway oedema prior to removing the endotracheal tube and this could be assessed by deflating the cuff and listening for a leak around the tube.

You are called to the ward to see this patient three hours after her surgery. She has acute respiratory distress with oxygen saturations of 78%. How would you proceed?

I would approach this problem with an airway, breathing and circulation approach. Whilst making an initial rapid assessment of the situation I would administer 100% oxygen via a non-rebreathing mask. The potential problems I need to consider are bleeding causing a haematoma around the surgical site, laryngeal oedema and damage to the recurrent laryngeal nerves.

The most likely cause is a haematoma and an urgent transfer back to theatre with senior anaesthetic and surgical personnel present should be organised. The incidence of post-operative haematoma is quoted as up to 4% and recent consensus guidelines have been published to guide the management. A post-thyroid surgery box should be present at the bedside, containing the guideline and the equipment to rapidly open the wound and evacuate a haematoma in situ according to a Skin exposure; Cut sutures; Open wound; Open muscles; Pack wound (SCOOP) approach. A return to theatre is indicated for careful wound exploration and cessation of any ongoing bleeding. Intravenous dexamethasone and tranexamic acid may be useful whilst arrangements for theatre are made.

A further rare cause of postoperative airway compromise in patients with a long-standing goitre is tracheomalacia. These patients should be reintubated quickly and are likely to require further management with a tracheostomy. This is more common following removal of longstanding, slow-growing goitres.

What are the consequences of damage to the recurrent laryngeal nerves?

The recurrent laryngeal nerves can be affected by compression, ischaemia or direct injury at the time of surgery. The injury can be unilateral or bilateral, and partial or complete. The recurrent laryngeal nerve supplies all the intrinsic muscles of the larynx except cricothyroid. Complete transection of the nerve results in the vocal cord on the affected side adopting a partially abducted or 'cadaveric' position. Partial nerve injury is potentially more worrying as the muscles controlling abduction of the cords are affected to a greater extent than the adductors and the cord lies in the midline. Bilateral partial

nerve injury can therefore result in the vocal cords overlapping in the midline producing complete airway obstruction.

Name another major postoperative complication of thyroid surgery aside from airway complications.

Inadvertent removal of parathyroid tissue at the time of thyroidectomy can produce hypocalcaemia. This manifests as muscle twitching, tingling around the mouth and tetany in extreme cases. Specific clinical signs can be elicited such as Trousseau's sign whereby carpopedal spasm is provoked by inflation of a cuff around the limb or Chvostek's sign where tapping over the course of the facial nerve produces twitching of the facial muscles. Corrected calcium levels should be checked routinely 12–24 hours after surgery and earlier if there is clinical suspicion of hypocalcaemia. Calcium supplementation can be oral in mild cases or intravenous if the corrected calcium is less than 2 mmol/L. Appropriate intravenous calcium supplementation is 10 ml of 10% calcium gluconate given over 3 to 5 minutes.

Further Reading

Cook T, Morgan P, Hersch P. Equal and opposite expert opinion. Airway obstruction caused by a retrosternal thyroid mass: Management and prospective international expert opinion, *Anaesthesia*. 2011; 66: 828–836.

Iliff H et al. Management of haematoma after thyroid surgery: Systematic review and multidisciplinary consensus guidelines from the Difficult Airway Society, The British Association of Endocrine and

Thyroid Surgeons and the British Association of Otorhinolaryngology, Head and Neck Surgeons, *Anaesthesia*. 2022; 77: 82–95.

Malhotra S, Sodhi V. Anaesthesia for thyroid and parathyroid surgery, *British Journal of Anaesthesia – Continuing Education in Anaesthesia, Critical Care and Pain*. 2007; 7 (2): 55–8.

Parry Z, Macnab R. Thyroid disease and thyroid surgery, *Anaesthesia and Intensive Care Medicine*. 2017; 18(10): 488–495.

1.3.11 Pheochromocytoma – Caroline SG Janes and Prakash Vadukul

A 45-year-old man was referred for investigation of resistant hypertension. Eventual blood testing and imaging undertaken by the hypertension clinic discover the presence of a large adrenal mass. He has been listed for excision of this mass by a laparoscopic approach.

What is resistant hypertension?

Blood pressure that is uncontrolled despite treatment with optimal, tolerated doses of an ACE inhibitor (or ARB) plus a calcium channel blocker plus a thiazide-like diuretic.

What are pheochromocytomas?

Pheochromocytomas may be found in 0.2–0.6% of patients with hypertension.

These are rare tumours of the adrenal medulla characterised by catecholamine secretion; adrenaline, noradrenaline and sometimes dopamine. Paraganglionomas are

neuroendocrine tumours originating from the extra-adrenal paraganglia and can also produce catecholamines.

These tumours are derived from chromaffin cells (phaeochromocytes) which in health are responsible for catecholamine release in response to stimulation.

The 10% rule referred to the previous thought that 10% of these tumours were bilateral, malignant, familial and occur in childhood. Now it is thought that up to 30% are malignant, 25% are extra-adrenal and 30% are familial. Around 10% of patients may be normotensive which may relate to receptor downregulation or desensitisation.

What conditions are associated with them?

These tumours are mostly sporadic but they can be associated with the multiple endocrine neoplasia syndromes 2A and 2B (thus having an association with medullary thyroid cancer and parathyroid adenomas). Neurofibromatosis and Von Hippel-Lindau disease are known associations.

How do they present?

Hypertension which is resistant to treatment is a common feature. A classical triad is often described including hypertension, headache and sweating. Other nonspecific symptoms resulting from sympathetic stimulation include palpitations (may be a manifestation of arrhythmias), panic attacks, nausea, visual disturbance and abdominal pain.

Many are found incidentally during imaging of the abdomen undertaken for other indications.

What testing is undertaken?

Biochemical testing is based on quantifying catecholamine breakdown products. Prior testing strategies involved arduous 24-hour urinary collection and analysis of levels of urinary catecholamines and vanillylmandelic acid. Current testing uses either serum or urinary free metanephrines or normetanephrines.

Imaging is typically undertaken after positive biochemistry and this may involve either CT or MRI scanning which have comparable sensitivity. Scintigraphy using radioactive meta-iodobenzylguanidine (MIBG) is sometimes used to further characterise the disease including the presence of extra-adrenal or metastatic disease. Adrenergic tissues demonstrate strong uptake of MIBG and this can be quantified using a detector.

What are the goals of preoperative optimisation?

Patients should undergo physiological stabilisation before surgical resection. Case reports describe intraoperative catecholamine crises and fatal hypertensive crises when these types of lesion are encountered unexpectedly. Indeed early surgical interventions for these tumours carried high risk of mortality due to the occurrence of stroke, cardiac failure and arrhythmia.

The key goals include control of blood pressure, optimisation of circulating volume, glycaemic control, dyselectrolytaemia management and assessment and optimisation of cardiac function.

Involvement of a multidisciplinary team including endocrinologists is important to ensure adequate biochemical control prior to embarking on surgical intervention.

Surgical intervention is unlikely to be an emergency; however, many of these tumours do have malignant potential and therefore a balance between optimisation and surgical expediency is required.

Patients may also present with altered glucose metabolism and this is again mediated by excess circulating catecholamines.

α -1 adrenoreceptor stimulation leads to increased glycogenolysis, α -2 leads to reduced insulin release, β -1 stimulation leads to increased lipolysis and β -2 stimulation leads to increased glucagon secretion and increased peripheral insulin resistance.

Patients are often significantly volume depleted due to chronic vasoconstriction leading to pressure natriuresis as well as the effects of excessive sweating and osmotic effects of hyperglycaemia.

How is preoperative blood pressure control approached?

Standard outpatient blood pressure management strategies will not be effective as drugs such as angiotensin converting enzyme inhibitors will not address the underlying pathology.

Treatment is initiated with α -blockade using drugs such as phenoxybenzamine or doxazosin.

Phenoxybenzamine is a long-acting, non-selective, non-competitive α -blocker which binds irreversibly to α -receptors. Its long duration of action means that it has implications for perioperative hypotension meaning that it is usually stopped 1–2 days before surgery. Due to its effect on α -2 receptors there may be undesirable sedative effects.

Doxazosin is a selective α -1 blocker which may lead to less effect on postoperative hypotension. A 2020 trial comparing the two drugs suggested similar efficacy of blood pressure control during surgery.

Calcium channel blockers may additionally be utilised as an adjunct in suboptimal control with alpha blockade alone.

In general, goals include symptom control (reduced sweating, anxiety, headaches) with consistent blood pressure readings below 160/90 mmHg. Typical treatment courses prior to surgery are generally a minimum of two weeks but may be prolonged for treatment of refractory hypertension.

Why is it important to start alpha blockade prior to beta blockade?

β -blockade may be desirable as this population are prone to catecholamine mediated dysrhythmias and many patients have symptomatic palpitations. Treatment with α -blockade may also lead to tachycardia.

Commencement of beta blockade prior to adequate α -blockade may lead to a hypertensive crisis. β 2-mediated vasodilatation is antagonised leading to unchecked α -agonism and vasoconstriction.

Typical β -blocker therapy involves the use of drugs such as atenolol, propranolol or metoprolol.

What other investigations would you consider before anaesthesia and surgery?

Patients with long-standing catecholamine excess are at risk of dysrhythmias and cardiac dysfunction. A 12-lead ECG may demonstrate ventricular hypertrophy or features of

strain or ischaemia. Echocardiography is an essential tool as most patients have a degree of diastolic dysfunction (heart failure with preserved ejection fraction) and a significant proportion may have systolic dysfunction (heart failure with reduced ejection fraction).

A full panel of bloods is warranted including urea and electrolytes; this population is at risk of renal impairment. At minimum a group and save is required given the potential for intraoperative bleeding and the need for perioperative transfusion.

What surgical approaches for phaeochromocytoma management are there?

Surgery may be open or laparoscopic depending on the location or size of the tumour. Open techniques may be required for larger tumours or those which are more invasive. Laparoscopic adrenalectomy is generally conducted in the lateral position with significant table breaks to facilitate adequate port placement as for laparoscopic approaches to nephrectomy.

What are the goals of anaesthesia for this surgery?

This can be challenging surgery and it is firstly essential that the patient is sufficiently medically optimised and that there is sufficient senior support present including personnel experienced in managing the demands of this type of surgery.

The key aspect of phaeochromocytoma surgery is to mitigate haemodynamic responses to anaesthesia and surgical interventions, clearly particularly during direct tumour handling.

Would you avoid any specific drugs?

Several drugs may potentially have adverse effects in this situation. Catecholamine increase can be a byproduct of histamine release, pre-synaptic release or reduced re-uptake. Thus, drugs such as desflurane, ketamine, morphine, pethidine, atracurium, ephedrine and metoclopramide may warrant care before use.

What monitoring devices would you want? Is there any role for cardiac output monitoring?

Arterial and central venous access are important. Arterial access is best sited prior to induction of anaesthesia given the risks of haemodynamic instability during intubation. Central venous access is appropriate due to the need for multiple infusions and the potential need for vasopressors after surgery.

Cardiac output monitoring may be desirable although there is little evidence to support its use in this type of surgery. It may be a useful modality in the patient with impaired cardiac function to guide fluid replacement. There is some evidence to suggest that cardiac output monitoring devices, for example LiDCo, may be beneficial to guide the treatment of vasoplegia in the perioperative setting.

Urinary catheterisation and temperature monitoring as for any major surgery is recommended.

What are the challenges of intraoperative management?

There are anaesthesia and surgical factors which can lead to catecholamine release.

Anaesthesia

- Drug triggers
- Tracheal intubation
- Coughing
- Pain

Surgical factors

- Incision
- Pneumoperitoneum
- Tumour handling

What specific management strategies are you aware of?

There is little formal RCT-based evidence to direct management of pheochromocytoma and thus strategy should revolve around personal experience as well as local and regional guidance.

For the management of pain and the responses to intubation remifentanyl is a good solution. With its rapid onset and favourable pharmacokinetic characteristics it can be rapidly titrated to effect to provide analgesia and obtund sympathetic responses to laryngoscopy. Clearly it will not affect the hypertension caused by physical excess of catecholamines during tumour manipulation. High dose fentanyl (up to 5 mcg/kg) has also been used to modify response to laryngoscopy.

Magnesium sulphate usage has increased significantly and is widely used in major surgery for its antinociceptive effects. Specific advantages in this situation include the inhibition of adrenal catecholamine release and reduced receptor sensitivity.

Dexmedetomidine has also been utilised. The central α -2 blockade has both analgesic properties but also leads to a reduction in circulating catecholamines.

What about specific blood pressure controlling treatments?

Tumour handling can lead to significant hypertension and dysrhythmias. Although surgical ligation of the adrenal vein will reduce catecholamine delivery to the circulation there may still be issues with severe hypertension afterwards. Thus pharmacological blood pressure control is an essential aspect of successful surgery as is communication between the surgical and anaesthesia teams.

Short-acting, titratable agents are preferable and as previously little direct evidence exists for superiority of one over the other. Well described approaches include:

Phentolamine

Short-acting, α -blockade resulting in vasodilation with doses of 1–2 mg. May demonstrate some tachyphylaxis and thus may be useful as a bolus dosing with other infusions.

Sodium nitroprusside (SNP)

Arteriolar vasodilation, rapid onset and offset. Typically used as an infusion with dose range from 0.5 to 1.5 mcg/kg/min to begin with up to 4 mcg/kg/min.

Glyceryl trinitrate

Similar action to SNP, more pronounced venodilation. Potentially preferable in those patients with ischaemic heart disease due to preservation of coronary perfusion. Dose range as an infusion from 10 to 200 mcg/min.

Esmolol

Short-acting, selective β -1 antagonism. May be beneficial in management of arrhythmia and tachycardia.

Surgery progresses well but following tumour removal there is hypotension. What might be happening and how can this be treated?

This type of surgery often sees variable blood pressure with the effects of tumour handling leading to rises and the titration of anaesthesia, analgesia and anti-hypertensives leading to hypotension. Fluids, titration of analgesia/anaesthesia and judicious vasopressors may be required.

Following tumour devascularisation there may be treatment resistant hypotension. This is thought to be multifactorial secondary to long-lasting α -blockade preoperatively, large drops in catecholamine concentration and receptor desensitisation from chronic catecholamine excess.

This can be addressed with fluid and vasopressors. Noradrenaline can be utilised to manage vasoplegia although mechanistically, vasopressin may be superior given the non-catecholamine driven effects of systemic vasoconstriction through V-1 receptors.

Some case reports also describe the use of hydroxocobalamin to address persistent post-procedure vasoplegia.

There may also be myocardial dysfunction. As with any major surgery it is essential to consider haemorrhage.

What level of postoperative care is needed?

Following uncomplicated surgery patients can be extubated. Ongoing vasopressor requirements mandate ongoing care in a high dependency setting. Invasive monitoring should be continued for 24–48 hours after surgery.

Hypoglycaemia may be apparent due to the loss of adverse metabolic effects of excess catecholamine secretion.

Steroid replacement is required after bilateral procedures.

How would you manage an unexpected pheochromocytoma?

Management principles for unexpected cases are the same as for elective scenarios. Acquiring assistance is essential and perioperative support on intensive care will be needed. Surgery should be stopped as soon as is both safe and feasible.

How would you manage a patient who is diagnosed with a pheochromocytoma during pregnancy?

There are two options for managing a pregnant patient with a pheochromocytoma. The tumour can either be excised during the second trimester or a simultaneous caesarean

section and tumour excision can be done in the third trimester. The overall mortality is about 17% and is highest with a normal vaginal delivery. Phenoxybenzamine and propranolol can be safely used during pregnancy for symptom control.

Further Reading

- Connor D, Boumpfrey S. Perioperative care of pheochromocytoma. *British Journal of Anaesthesia Education*. 2016;16(5): 153–158.
- Giustini AJ, Rowe EV, Perez FD, Mihm FG. Hydroxocobalamin to treat refractory vasoplegia following pheochromocytoma resection in a child. *Anaesthesia Reports*. 2022 Dec 11;10(2): e12201.
- Nölting S, Bechmann N, Taieb D, Beuschlein F, Fassnacht M, Kroiss M, Eisenhofer G, Grossman A, Pacak K. Personalized management of pheochromocytoma and paraganglioma. *Endocrine Reviews*. 2022;43(2): 199–239.
- Pace N, Buttigieg M. Phaeochromocytoma. *BJA CEPD Reviews*. 2003; 3(1): 20–23.
- Sasidharan P, Johnston I. World Federation of Societies of Anaesthesiologists: Phaeochromocytoma: Perioperative Management. Available from: <https://resources.wfsahq.org/atotw/phaeochromocytoma-perioperative-management/> (Accessed 15 December 2022).

Gastrointestinal

1.4.1 Enteral and Parenteral Nutrition – Caroline SG Janes and Prakash Vadukul

A 55-year-old man requires admission to intensive care following deterioration with multiple organ failure from acute pancreatitis. He requires mechanical ventilation due to increasing oxygen requirements. A nasogastric tube is inserted to facilitate feeding.

What is enteral nutrition?

Enteral nutrition (EN) is that which is delivered to and uses the innate function of the gastrointestinal system. The usual route of nutritional delivery is through volitional intake of food orally followed by chewing, swallowing and digestion.

What are the benefits of enteral nutrition?

There are various benefits to EN and this should be the first line of nutritional delivery in all patients.

Wherever possible patients should eat normally; however, during periods of critical illness this may require input from dieticians and speech and language therapists to provide guidance on what the patient requires and whether normal eating is safe.

Additional benefits include the maintenance of gut integrity and subsequent protection against bacterial translocation.

Liquid enteral feeding buffers gastric acid, increases mucosal blood flow and induces the secretion of protective prostaglandins and mucous. This reduces the risk of stress ulceration and much of the evidence suggests that with successful enteral feeding it is safe to discontinue specific therapies for ulcer prophylaxis such as proton pump inhibitors.

What routes are available to facilitate EN?

The oral route is preferred but there are various other delivery routes available depending on specific patient requirements:

- Nasogastric – most common. Orogastic tubes can also be utilised for short-term periods in patients with specific contraindications to NG tube placement e.g. base of skull injury.
- Nasojejunal – post pyloric feeding is not routinely required but may be needed in situations where there is gastric outlet obstruction. This may be the case in situations such as pancreatitis. This route can be more challenging to facilitate as tubes often require endoscopic guidance for correct placement.

- PEG (percutaneous endoscopic gastrostomy) – this route may be utilised where there is an anticipated failure of feeding via the aerodigestive tract. This may be the situation in chronic neuromuscular disorders where this is a risk of aspiration or in situations such as head and neck malignancy.

Can you tell me about recommendations before starting feed via the nasogastric route?

Due to the incidence of feeding through inappropriately placed NG tubes, the National Patient Safety Agency set out criteria for checking tube placement to prevent further occurrence of these ‘Never Events’.

The typical process involves testing gastric aspirates and commencing feed if the pH is in the safe range of 1 to 5.5. If this is not possible or the aspirate falls outside of this range, radiological confirmation is required.

What are the radiological criteria for safe nasogastric tube feeding?

- The tube should follow the oesophagus and avoid the bronchial contours.
- The tube should clearly bisect the carina or the bronchi.
- The tube should cross the diaphragm in the midline.
- The tip of the tube should be clearly visible below the left hemidiaphragm.

What are the complications of enteral nutrition?

Mechanical problems

- Tube obstruction
- Tube displacement
- Discomfort
- Tube related ulceration

Metabolic problems

- Dehydration or over hydration
- Hyperglycaemia
- Dyselectrolytaemia

Gastrointestinal

- Nausea
- Vomiting
- Diarrhoea (be mindful of the risk of infective diarrhoea e.g. *C.difficile*)
- Bloating
- Reflux
- Potential risk of aspiration and subsequent pneumonia

What is the approach to nutrition during critical illness?

The gut and in particular the small bowel are metabolically active and thus sensitive to critical illness where tissue oxygen delivery is reduced. With feeding, the tissues of the gastrointestinal tract have increased metabolic and oxygen demand. In critical illness this

may lead to an imbalance between supply and demand particularly in states requiring high doses of vasopressors or inotropic support. This may lead to functional ischaemia and lead to compromised gut integrity.

Guidance from the American Society for Parenteral and Enteral Nutrition (ASPEN) and the European Society for Clinical Nutrition and Metabolism (ESPEN) suggests EN should be initiated during the early stages of critical care admission and ideally within 24–48 hours.

ASPEN and ESPEN suggest in states where there is high vasopressor demand or need for inotropic support that EN be started at low doses. During active resuscitation or haemodynamic instability feed should be held.

Low dose vasopressor support is, however, not a contraindication to feeding and thus should be instituted. Most guidance advocates for feeding within the first week of critical illness.

What is trophic feeding?

This is a method of minimal rate feed (10–20 ml/hr of NG feed) which may mitigate some of the issues with feed intolerance during periods of haemodynamic instability or shock. Purported benefits include preservation of intestinal integrity, preservation of cell junctions and reducing risk of bacterial translocation.

What do you know about the adequacy of EN?

This may vary between departments and there is a lack of evidence. Tolerance of feeding can be assessed clinically (with attention to vomiting, abdominal distension and bowel sounds) and using gastric residual volumes (GRV). ASPEN suggests continuing feeding for residual volumes <500 ml at 6 hours albeit at a lower rate.

Other strategies may include the use of 4-hourly aspirates and continuing feeding with GRVs of 200–250 ml with adjusted feeding rates.

Prokinetics may be utilised in situations where there are persistently high GRVs (>250 ml at 4 hours). This includes metoclopramide 10 mg IV 8-hourly and erythromycin 250 mg IV 6-hourly.

Supplemental TPN may be considered if a period of trialling these measures fails, generally considered to be five or more days of inadequate enteral nutrition. If there are mechanical issues, for example gastric outlet obstruction, then post pyloric feeding can also be considered.

What is the impact of propofol? And can you tell me about overfeeding?

Propofol is formulated as lipid-water emulsion with refined soya-bean oil and purified egg phosphatide and thus carries significant calorific content, which requires attention when calculating dietary requirements for patients. 1% formulations contain around 1 kcal/ml.

Overfeeding can be problematic and may be a possibility without careful consideration. It is associated with hyperglycaemia, hepatic steatosis, hypertriglyceridaemia and excess carbon dioxide production (which may be particularly problematic for those with respiratory disease).

What nutritional supplements in critical care do you know about?

Amino acid demand is increased during critical illness and additional replacement has been studied. Glutamine and arginine have been investigated with some potential positive effects although there are no recommendations favouring routine supplementation.

What are the constituents of enteral feeds?

Feeds will provide water and some form of carbohydrate, protein and fat. Typical sources of these may include maltodextrin (carbohydrate), soy, casein (protein) and different oil types e.g. sunflower/corn/canola (fat).

Within these formulations there will also be suspensions of vitamins and minerals. Feeds may be tailored to suit particular clinical needs including low volume (for fluid-restricted patients), higher fibre or different electrolyte concentrations for example.

- Liver disease – altered amino acid contents to protect against encephalopathy
- Renal disease – low phosphate, low potassium, concentrated to 2 kcal/ml for fluid restriction
- Respiratory disease – reduced fat content to reduce CO₂ production.

Can you tell me the contraindications to EN?

These are usually affecting patients with gastrointestinal pathology including:

- Obstruction
- Perforation
- Mesenteric ischaemia
- Major gastrointestinal bleeding
- Abdominal compartment syndrome.

What are the indications for total parenteral nutrition?

This will broadly relate to the contraindications for enteral nutrition. More specifically the requirement for TPN is as a result of intestinal failure.

Therefore the indications will be:

- Where enteral nutrition is truly contraindicated
- Where enteral nutrition has failed despite undertaking other efforts or is deemed insufficient for the specific needs of the patient
- Short bowel syndrome leads to a variety of nutritional deficiencies and may thus require prolonged nutritional support.

For the critically ill patient there is a lack of consensus regarding when to start TPN. Recommendations focus on enteral feeding strategies and when these fail considering supplementing intake with PN. Studies have shown that there is no benefit to early initiation of PN in those at risk of malnutrition. With persistent failure to institute adequate EN, supplemental PN can be delivered alongside EN.

How might TPN be delivered?

The decision to commence TPN is generally not an emergency and therefore optimisation of access can be sought beforehand. The expected duration of TPN utilisation usually dictates the type of access desired.

TPN is significantly hypertonic and thus requires administration into venous sites with high flow rates and lower susceptibility to thrombophlebitis.

TPN can be specifically formulated to lower osmolalities (<850 mOsmol/L) thus enabling shorter-term delivery into smaller veins through small peripheral catheters.

For shorter-term periods of up to 30 days TPN can be delivered via peripherally inserted central catheters (PICC) or non-tunnelled central venous catheters. Some guidance advocates for the use of PICCs for up to 3 months of TPN administration.

For longer-term TPN duration (>30 days/>3 months) tunnelled central venous access is the recommended route of delivery. This process should be led by local nutrition teams.

What does TPN contain and how is it formulated?

TPN can often be commenced using generic, starter formulations prior to dietician or nutritional specialist involvement. TPN bags can then be formulated to specific patient requirements.

Given the risk of infection TPN requires careful handling and requires sterile production techniques.

As for enteral formulations there will broadly be amounts of carbohydrate, fat and protein with added elements, electrolytes and vitamins.

Carbohydrate are supplied in the form of glucose at concentrations of 40–70%.

Lipids have a calorie load and provide fatty acids which have important physiological functions including cell membrane synthesis and function with additional roles in immune function. Lipid emulsions form the basis of lipid delivery and different blends are available. Broadly the fats available include soybean oil, coconut oil, olive oil and fish oils.

Protein delivery involves administration of amino acids. Essential amino acids (those which are not made in the body) must be added to parental nutrition formulations.

Trace elements such as zinc, copper, selenium can also be added to TPN formulations. Vitamins such as thiamine are important components as deficiencies can lead to neurological dysfunction.

Electrolytes and water are the final components.

What are the complications of TPN?

These can be split into those related to the formulated TPN itself and those related to the access required to deliver it.

Those related to the TPN itself:

- Volume overload
- Electrolyte disturbances
- Cholestasis
- Intestinal atrophy
- Hyperglycaemia – high intravenous glucose delivery combined with a relative insulin-resistance during critical illness.

Those related to access:

- Line infection – this warrants meticulous attention in all patients but particularly with regard to TPN. TPN should be administered through a dedicated lumen and disconnections should be minimised
- Thrombophlebitis can occur with delivery into peripheral vein.

Further Reading

Bratanow S, Brown S. Nutrition in the critically ill. Update in Anaesthesia. Available from <https://resources.wfsahq.org/wp-content/uploads/uia28-Nutrition-in-the-critically-ill.pdf> (Accessed 30 December 2022).

Chowdhury, R, Lobaz S. Nutrition in critical care. *British Journal of Anaesthesia Education*. 2019; 19(3): 90–95.

Macdonald K, Page K, Brown L, Bryden D. Parenteral nutrition in critical care. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2013; 13(1): 1–5.

NHS Improvement. Initial placement checks for nasogastric and orogastric tubes. Available from: www.england.nhs.uk/wp-content/uploads/2016/07/Resource_set_-_Initial_placement_checks_for_NG_tubes_1.pdf (Accessed 30 December 2022).

Piton G, Gouge AL, Boisramé-Helms J, Anguel N, Argaud L, Asfar P, et al. Factors associated with acute mesenteric ischemia among critically ill ventilated patients with shock: A post hoc analysis of the NUTRIREA2 trial. *Intensive Care Medicine*. 2022; 48(4): 458–466.

1.4.2 Nutritional Requirements and Malnutrition –

Caroline SG Janes and Prakash Vadukul

A 24-year-old man with a BMI of 18 and a background of inflammatory bowel disease is admitted with a severe flareup of his disease. He is being worked up for potential emergency sub-total colectomy as an inpatient.

What are the typical daily nutritional requirements for an adult patient?

Broadly speaking humans have daily requirements for a quantity of water, carbohydrate, fat, protein and electrolytes to maintain bodily function and homeostasis (Table 1.4.2).

What are micro and macronutrients?

Macronutrients refer to fats, carbohydrates and protein.

Micronutrients refer to vitamins, minerals and trace elements.

Table 1.4.2 Daily adult nutritional requirements

Water	30 ml/kg
Energy	25 kcal/day
Carbohydrate	2 g/kg
Protein	0.8–1.2 g/kg
Fat	1 g/kg
Sodium	1–2 mmol/kg
Potassium	0.8–1 mmol/kg
Calcium	0.1 mmol/kg
Magnesium	0.1 mmol/kg
Phosphate	0.2–0.5 mmol/kg

What do you know about trace elements?

These are ions such as iron, zinc, copper and selenium. They play important roles for enzyme function and many have important antioxidant properties which contribute to immune system function.

What is malnutrition and why is it important?

This is a state caused by an imbalance of nutrients (which can be related to insufficiency or inadequacy) which can lead to tissue and body dysfunction. Obese patients may present with an excess of macronutrients but a lack of important micronutrients.

It is a common phenomenon, being present in up to 40% of patients on admission to hospital.

It is a risk factor for increased morbidity, mortality, length of stay, high readmission rates, lower quality of life and impaired recovery after surgical intervention as well as delayed wound healing.

In the critical care population, malnourished patients are more prone to complications. The sequelae of critical care, particularly muscle loss and diaphragm weakness, are amplified in this group. Thus there is an increased likelihood of prolonged weaning from mechanical ventilation with the inherent risks therefore of further ventilator-acquired pneumonia.

How is the body effected by malnutrition?

- Central nervous system – impaired cognition, fatigue, weakness
- Musculoskeletal – reduced muscle mass, reduced bone mass, impaired wound healing
- Cardiovascular – reduced cardiac output, arrhythmia (secondary to electrolyte disturbance)
- Respiratory – reduced respiratory muscle strength and function, increased risk of pneumonia
- Renal – reduced glomerular filtration rate, high total body water
- Gastrointestinal – gut atrophy, increased bacterial translocation, oesophagitis
- Haematological – leucopenia, anaemia, pancytopenia
- Vitamin deficiencies – can produce a milieu of effects related to specific deficiency syndromes
- Electrolyte disturbance – hypokalaemia, hypocalcaemia.

Which populations are at risk of malnutrition?

These can be split into the following categories

Patient characteristics, including:

- Elderly (reduced volitional intake)
- Malignancy
- Respiratory disease (particularly COPD)
- Inflammatory bowel disease
- Social factors (homelessness, alcohol/substance abuse, neglect)
- Anorexia, bulimia, depression

- Patients with head and neck malignancy
- Hyperemesis

Acute presentations, including:

- Trauma
- Burns
- Sepsis
- Critical care admission

Surgical factors, including:

- High output stoma
- Gastrointestinal fistula
- Bowel obstruction

How can nutritional state be assessed?

There are a number of scoring systems and assessment tools for assessing patient nutrition; however, history taking and examination are useful tools. The Subjective Global Assessment tool combines history and examination to identify risk of malnutrition. History taking focusses on the loss of weight, the inadequacy of diet and on symptoms such as nausea, vomiting and diarrhoea. Examination may reveal cachexia, oedema or dehydration.

Haematological and biochemical investigations may show anaemia, electrolyte disturbances (low potassium, phosphate, magnesium) and low albumin levels. These may be confounded by acute illness (albumin reduction as an acute phase protein during illness).

Anthropometric assessments are less commonly utilised as they tend to be unreliable in the context of oedema and fluid shifts. They may involve measurements such as skin-fold thickness and mid arm circumference.

Body mass index ($\text{mass [kg]} / \text{height}^2 [\text{m}^2]$) is a simple measurement and has been demonstrated to be linked to mortality in the critically ill population. BMI < 18.5 is classed as underweight.

Formal scoring systems are routinely utilised in the United Kingdom. NICE recommends screening on admission to hospital e.g. the Malnutrition Universal Screen Tool (MUST). MUST uses BMI, weight loss and the presence of acute illness potentially causing reduced nutritional intake to identify those who are malnourished or at risk.

Other tools include the Nutritional Risk Screening (NRS-2002) and the Nutrition Risk in the Critically Ill Score (NUTRIC).

Ultimately nutritional management is a multidisciplinary process requiring input from dieticians to enable prompt identification and management of those at risk of malnutrition.

How can energy requirements be calculated?

In states of acute illness or following surgical stress there is increased energy expenditure and it is therefore important to meet these needs to prevent catabolism and muscle breakdown.

Indirect calorimetry is suggested as the most accurate method. This technique measures oxygen consumption and carbon dioxide production. In practice this is less

practical to perform requiring measurements from ventilators or pulmonary artery catheters. The technology for this may become more prominent in the future.

Equations such as the Harris-Benedict equation are sometimes utilised in the critical care environment. This tool estimates the basal metabolic rate in a calories/day format. Sex, weight, height and age are used during these calculations.

What is refeeding syndrome? How might it be mitigated?

After a period of starvation, typically thought to be five days or more, physiological responses can lead to problematic metabolic and electrolyte disturbances on resumption of diet.

During periods of starvation there is a switch to fat metabolism from regular carbohydrate metabolism. There is an associated drop in endogenous insulin. Stores of electrolytes are depleted with inadequate oral intake. Although there may be adequate serum concentrations, there is likely to be a total body deficit with low intracellular levels.

With dietary resumption insulin levels sharply rise. As a consequence there is glycogen, fat and protein synthesis. These processes require phosphate, potassium and phosphate. Due to the intracellular shifts of these electrolytes, serum levels can decline precipitously. Hypophosphataemia is the commonest biochemical feature of refeeding syndrome. Severe hypophosphataemia can result in confusion, delirium, seizures, respiratory failure and cardiac failure.

Monitoring is an essential aspect as is the replenishment of these electrolytes and vitamins such as thiamine. NICE guidance recommends slow commencement of nutrition usually at 50% of estimated energy requirements with slow increments to full feeding over the course of days increasing by 200–400 kcal/day. In severe cases 5–10 kcal/kg/day may be the starting rate of feed.

What special situations are there regarding nutritional requirements during critical illness?

- Burns – this type of patient may require up to a full doubling of calculated energy requirements due to profoundly inflammatory and catabolic state. This is more so with larger burns >30% total body surface area
- Sepsis – patients may require up to 9% more nutrition
- Following surgery or trauma – up to 6% more nutrition.

What is the relevance of nutrition to surgical outcomes?

Preoperative malnutrition leads to increased susceptibility to infection, impaired wound healing and increased length of stay.

What can you tell me about prehabilitation and the relationship to nutrition?

This is a multidisciplinary strategy which aims to optimise preoperative fitness via physiological and functional reserve, and physical condition, prior to embarking on surgical interventions. There are physical as well as psychosocial aspects.

Prehabilitation programmes include nutritional support aiming at increasing muscle mass as well as exercise and lifestyle support, for example smoking cessation.

Tell me about the stress response to surgery.

This is the term used to describe the physiological changes in response to surgical trauma. There are metabolic, hormonal and haematological changes which lead to an increase in oxygen consumption and a related increase in cardiac output. Energy requirements are increased and protein synthesis is required as part of the acute response to ensure tissue healing, immune system function and recovery.

The requirement for energy and protein leads to gluconeogenesis using lean tissues such as muscle as a substrate. There is a state of catabolism leading to wasting of lean tissues.

Why is nutrition important to manage the surgical stress response?

Physiological optimisation is essential to ensure that the ability to maintain oxygen delivery for organ function is preserved.

Nutritional optimisation plays a role in ensuring adequate lean body mass and energy reserves to adequately mitigate the stress response and meet the increased demand created by surgical stress while ensuring overall physiological stability. Without adequate nutritional reserves, oxygen delivery may be compromised leading to the increased risk of organ dysfunction and poor healing seen in malnourished states.

Can you outline an approach to anaesthesia for the malnourished patient?

Care should be taken to optimise the patient preoperatively as far as possible with attention paid to electrolyte and fluid deficiencies.

Pre-induction, consider the need for adjustments to dosage of induction agents. Additionally patients with malnutrition may have paradoxical slowed gastric emptying. Therefore consider the need for rapid sequence induction.

Intraoperative attention towards maintenance of normothermia is important as this patient population is at risk of heat loss due to reduced fat stores and higher body surface area. Cardiac output monitoring may be beneficial in the patient with cardiac dysfunction.

Depending on the severity of malnutrition there may be difficulty with extubation due to respiratory muscle fatigue and thus care should be taken to ensure reversal of neuromuscular blocking agents with monitoring throughout anaesthesia.

The patient deteriorates and requires emergency surgery due to refractory colitis. Intraoperative blood gas analysis reveals a profound acidosis.

What might be the cause?

As with any critically ill patient a structured approach is essential. In this situation it would be important to assess in particular for any respiratory or cardiovascular compromise. Catheterisation with observation of urine output is essential to assess for renal dysfunction.

Analysis of the anion gap may be beneficial. In the starved/malnourished patient it is important to consider ketoacidosis. This results in a high anion gap metabolic acidosis. Blood ketone measurement would enable diagnosis.

Hepatic glycogen stores are typically exhausted at 12–24 hours and there is a subsequent production of ketones as part of fatty acid metabolism for energy. Treatment involves administration of glucose to direct normal energy utilisation.

Further Reading

Bratanow S, Brown S. Nutrition in the critically ill. Update in Anaesthesia. Available from <https://resources.wfsahq.org/wp-content/uploads/uia28-Nutrition-in-the-critically-ill.pdf> (Accessed 30 December 2022)

Chowdhury R, Lobaz S. Nutrition in critical care. *British Journal of Anaesthesia Education*. 2019; 19(3):90–95.

Edwards S. Anaesthetising the malnourished patient. Update in Anaesthesia. 2016; 31:31–37. Available from [https://resources.wfsahq.org/wp-content/uploads/uia31-](https://resources.wfsahq.org/wp-content/uploads/uia31-Anaesthetising-the-malnourished-patient.pdf)

[Anaesthetising-the-malnourished-patient.pdf](#) (Accessed 20 December 2022).

Gillis C, Wischmeyer PE. Pre-operative nutrition and the elective surgical patient: Why, how and what? *Anaesthesia*. 2019; 74: 27–35.

National Collaborating Centre for Acute Care. Nutrition support in adults. Oral nutrition support, enteral tube feeding and parenteral nutrition. February 2006. Available from www.nice.org.uk/guidance/cg32/evidence/full-guideline-194889853 (Accessed 21 December 2022).

1.4.3 Postoperative Nausea and Vomiting (PONV) – Alison J Brewer

We are going to talk about postoperative nausea and vomiting. Can you tell me which groups of patients are more likely to suffer from this complication?

Be systematic with your opening statement.

There are patient, surgical, anaesthetic and pathological risk factors.

Patient factors include:

- Female gender (2–4 times more likely than males)
- Menstruation (during the luteal phase)
- Pregnancy (especially the first trimester)
- Obesity
- Younger age
- Smoking decreases the incidence
- Past history or travel sickness.

Surgical factors include:

- Type of surgery (especially strabismus, middle ear, intracranial, intra-abdominal and gynaecological surgery)
- Prolonged surgery.

Tell me about the anaesthetic and pathological risk factors.

Anaesthetic factors include:

- Drugs (nitrous oxide, opioids and inhalational agents)
- Prolonged anaesthesia and surgery
- Spinal anaesthesia and the associated hypotension also increase the risk
- Dehydration increases risk
- Gastric dilatation increases the likelihood of nausea and vomiting.

Pathological risk factors include:

- Intestinal obstruction and gastric stasis
- Hypoglycaemia
- Hypoxia
- Uraemia.

How can PONV be prevented?

There is a lot to cover here so again, be systematic. Classify as pre-, intra- and postoperative prevention.

There is now an emphasis on multimodal methods in the prophylaxis against PONV particularly in patients who are at risk. Preoperative scoring systems can help identify these patients.

Preoperative dehydration should be addressed and anxiety minimised. Intraoperatively, patients at high risk of PONV should have an anaesthetic that minimises the risk of nausea and vomiting. They should have propofol as an induction agent and nitrous oxide should be avoided. A total intravenous anaesthetic (TIVA) should be considered. The use of neostigmine should be kept to a minimum, as should sympathomimetics. Pain should be avoided by using opiate-sparing techniques. I would use paracetamol, diclofenac and local anaesthetic techniques in preference to opioids.

Classify the drugs used to prevent postoperative nausea and vomiting?

In our hospital the most common drugs used for prevention are cyclizine, ondansetron and dexamethasone.

More widely, the classes of drugs used to treat nausea and vomiting are:

- Antihistamines
- Antimuscarinics
- Antidopaminergics
- Antiserotoninergics
- Cannabinoids
- Corticosteroids.

They can be classified by site of action.

Outline the sites of action of antiemetics.

Peripherally there are visceral afferents in the bowel wall and myenteric plexus and these are mediated by serotonin (5-HT₃), where drugs such as ondansetron and granisetron work. The afferents neurons relay to the chemoreceptor trigger zone (CTZ). The CTZ, in the medulla in the area postrema on the floor of the fourth ventricle lies outside the blood-brain barrier and receives afferents from the vestibular apparatus and certain drugs such as opiates, volatiles and sympathomimetic drugs. Dopamine receptor antagonists act here to stimulate the CTZ. NK-1 receptors are also abundant at this site.

There is a vestibular input to the vomiting centre and anticholinergic drugs, (such as hyoscine) and antihistamine drugs (such as cyclizine) act here. The vomiting centre is the site of central integration and lies in the reticular formation in the medulla oblongata. It receives afferents from the CTZ, gut and the cerebral cortex. This area contains

muscarinic and histamine receptors and therefore drugs which act at the vestibular apparatus also work here.

Some drugs do not have a defined site of action. These include propofol, whose antiemetic actions are thought to be due to action on the CTZ. Corticosteroids, such as dexamethasone have an unknown site of action, but may be due to a reduced turnover of 5-HT₃, or a decreased permeability of the blood–brain barrier. Cannabinoids, such as nabilone are thought to act at the CTZ, but receptors CB-1 and CB-2 have been found and it may exert the antiemetic effects via these sites.

You mentioned that the chemoreceptor trigger zone is outside the blood–brain barrier. Is there any benefit to this location?

Yes, this is so that the area can be directly exposed to blood borne chemicals and response to these is not delayed. Some emetogenic agents cannot penetrate the blood–brain barrier (i.e. quaternary amines).

Can you tell me which antiemetics you would use and why?

If you have departmental guidelines or protocols, it makes sense to quote them.

We have departmental guidelines whereby patients are preoperatively assessed and graded into low, moderate or high risk for nausea and vomiting. In low-risk patients no specific antiemetics are used, for those in a moderate risk group, intravenous cyclizine 50 mg is administered at the induction of anaesthesia and the avoidance of emetogenic drugs and techniques. In high-risk patients, multimodal therapy is used with intravenous ondansetron 4 mg and cyclizine 50 mg being given, with consideration to using dexamethasone 8 mg. Other techniques, such as using TIVA and opioid-sparing regional techniques can be considered.

Can you tell me the side effects of these drugs?

Antimuscarinic drugs have an antisialogogue effect so dry mouth and eyes are common. They also cause sedation, amnesia and can precipitate a central anticholinergic syndrome. Glycopyrronium is the only drug that does not cause these central side effects as is a quaternary amine and does not cross the blood–brain barrier.

Antihistamines can have an anticholinergic effect and cause tachycardia post-injection.

Antidopaminergic drugs can cause dystonic and extra-pyramidal effects. They can rarely precipitate the neuroleptic malignant syndrome. The commonest side effects of ondansetron are headache, feeling hot and constipation.

What about the other drugs such as corticosteroids and cannabinoids?

Corticosteroids have few side effects when used as a one off, but long-term use can cause steroid psychosis and metabolic disturbances such as fluid retention, hypokalaemia and hyperglycaemia. Cannabinoids cause sedation and psychosis and their use is usually limited to patients receiving chemotherapy.

Why is a combination of drugs used?

This improves the efficacy of the drugs over monotherapy as the cause of nausea and vomiting is likely to be multifactorial and using drugs that act at different sites is likely to

have the best outcome. The number needed to treat is about five in the best multimodal therapy. However, this does vary according to the underlying rate of postoperative nausea and vomiting in the patient and surgical risk group. For example, the number needed to treat to prevent PONV in gynaecological surgery is much lower than in general surgery.

What is meant by number needed to treat (NNT)?

This is the number of patients that need to be treated to have a reduction in symptoms. It gives an indication as to the size of the treatment effect and is calculated by $1/\text{absolute risk reduction}$.

Do you know of any non-pharmacological ways of reducing nausea and vomiting?

Ginger is thought to help with nausea and vomiting, as is hypnotherapy. Acupuncture at the P6 point distal to the wrist crease between the flexor carpi radialis and palmaris longus tendons reduces the incidence of vomiting.

What are the physiological mechanisms of vomiting?

Vomiting is the retrograde passage of gastric contents through the mouth and in order for this to happen the body must undergo a sequence of events to prevent aspiration from happening. The afferent limb of the reflex of vomiting is coordinated in the vomiting centre. The efferent limb has motor output via the cranial nerves to the upper GI tract and through the spinal nerves to the diaphragm and abdominal muscles. A sensation of nausea is experienced followed by sympathetic activity, such as hyperventilation, sweating, peripheral vasoconstriction and tachycardia. Salivation is as a result of parasympathetic activity. The glottis closes and the breath is held in mid-inspiration. Vagal impulses cause a relaxation of the proximal stomach and then a retrograde giant contraction causing small bowel contents to be forced into the stomach. Expulsion is preceded by retching in many cases and the oesophageal sphincters and diaphragm relax allowing the gastric contents to be expelled.

Why do we want to reduce the incidence of nausea and vomiting in patients?

PONV is the most common side effect following anaesthesia for a surgical procedure, accounting for up to 30% of patients. Apart from making the patient feel awful it can cause further morbidity. The patient is at risk of aspiration, especially if drowsy following surgery. They are also at risk of dehydration and electrolyte imbalance. The act of vomiting can increase the intracranial and intraocular pressure as well as worsening pain and increasing the risk of wound dehiscence and hernia.

Tell me about the electrolyte imbalances.

As a result of protracted vomiting, gastric contents are lost and this contains hydrogen, potassium, sodium ions and water.

What physiological mechanisms compensate for this loss?

There is renal compensation to restore the pH. To compensate for the volume loss there is aldosterone release, in response to the sodium and extracellular fluid depletion, to cause sodium and water retention in exchange for potassium and hydrogen ions. A metabolic alkalosis develops in vomiting.

1.4.4 Oesophageal Reflux – Michael B Clarke

Describe the anatomy of the oesophagus.

The oesophagus is a muscular tube connecting the pharynx to the stomach. The upper third of the oesophagus consists of an outer longitudinal and an inner circular layer of striated muscle. The lower two thirds consist of smooth muscle. The inner lumen consists of stratified squamous epithelium.

There are two oesophageal sphincters: the cricopharyngeus muscle acts as the functional upper oesophageal sphincter. It has a high resting intraluminal pressure (6.7–13.3 kPa); the lower oesophageal sphincter is a functional entity rather than an anatomical one. It results from thickened, tonically contracted smooth muscle at the lowest 2–4 cm of the oesophagus. It has a resting pressure 2–3.3 kPa above the gastric pressure, which prevents gastro-oesophageal reflux.

What is the ‘barrier pressure’ and what is its significance?

Barrier pressure is the lower oesophageal sphincter pressure minus the intragastric pressure. The significance is that the lower oesophageal sphincter pressure is reduced by pregnancy and drugs such as atropine and suxamethonium and that the intragastric pressure is increased in the fed state. When one encroaches on the other, barrier pressure fails and reflux is possible.

Describe the anatomy of the stomach.

Functionally, the stomach is made up of the fundus, the body and the antrum. The wall of the stomach consists of four layers: serous, muscular, submucous and mucous. There are three layers of visceral muscle fibres: longitudinal, circular and oblique. Each muscle layer forms a syncytium acting as a unit. Between the stomach and the duodenum is the pyloric sphincter, a junction formed by thickened circular smooth muscle.

The stomach has an intrinsic and extrinsic nerve supply. The extrinsic nerve supply is from the sympathetic and parasympathetic nervous systems. The sympathetic supply (via the coeliac plexus) inhibits motility. The parasympathetic supply (via the vagus nerve) stimulates motility.

The intrinsic nerve supply is responsible for peristalsis and is formed by the Meissner’s plexus which is submucosal, and the Auerbach’s plexus which lies between the circular and longitudinal muscle layers of the stomach.

How is gastric emptying normally controlled and what are the causes of delayed gastric emptying?

With an open question, an opening simple classification will aid your answer and impress the examiner.

The physical state and chemical composition of a substance affects the speed at which it is emptied from the stomach. Liquids empty more rapidly than solids. The rate of emptying of solids depends on the rate at which chyme is broken down into smaller particles. Increased gastric volume produces distension which provokes vagal reflexes leading to increased gastric emptying. Both gastric distension and high protein content of food stimulate gastrin secretion which enhances gastric emptying. Nutrients in the duodenum activate chemoreceptors which inhibit gastric emptying, allowing time for further digestion and absorption in the small intestine. Hypertonicity, fatty acids and hydrogen ions activate the secretion of cholecystokinin, secretin and gastric inhibitory peptide, which inhibit gastric emptying.

Many factors cause delayed gastric emptying and can be classified as physiological, pathological and pharmacological.

Physiological causes include:

- Pain
- Anxiety
- Pregnancy
- Age.

Pathological causes include:

- Gastrointestinal obstruction
- Electrolyte abnormality
- Diabetes
- Gastritis
- Raised intracranial pressure
- Migraine.

Pharmacological causes include:

- Opioids
- Anticholinergics
- Sympathomimetics
- Dopaminergics
- Alcohol.

What is gastric acid and what stimulates its production?

To help you answer the second part of this question a simple diagram of a parietal cell including the three main receptors and the hydrogen ion pump will impress the examiner and will aid you if a further question about drug actions is asked.

Gastric secretions include hydrochloric acid, pepsin, gastrin, mucus and intrinsic factor. The parietal cells secrete hydrochloric acid and intrinsic factor. These cells are located in the body and fundus of the stomach. A pH of 1 to 1.5 is needed for optimal pepsin activity as well as providing a degree of antimicrobial activity. Pepsin initiates protein digestion, intrinsic factor is necessary for vitamin B12 absorption in the terminal ileum and mucus is essential for protection of the mucosal cells

The three most important factors that act on the parietal cell to stimulate hydrochloric acid production are histamine (via H₂ receptors), acetylcholine (via M₁

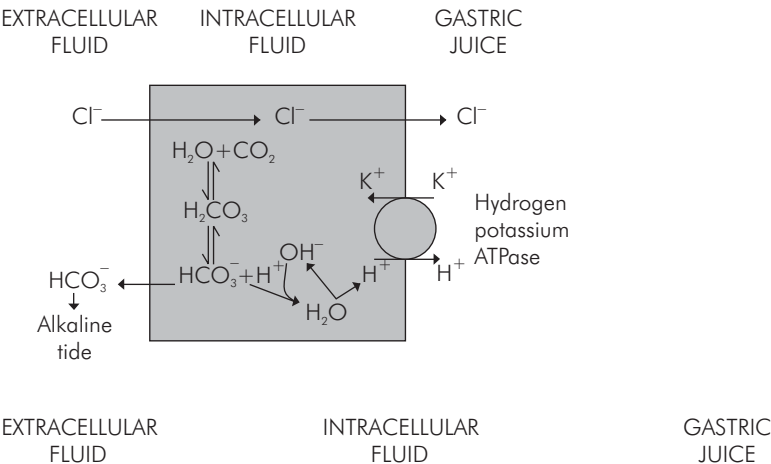


Figure 1.4.4.1 GI10 Acid production in parietal cells.
 Reproduced with permission from Smith, T., Pinnock, C. and Lin, T. 2009. *Fundamentals of Anaesthesia*. Cambridge: Cambridge University Press. © Cambridge University Press 2009.

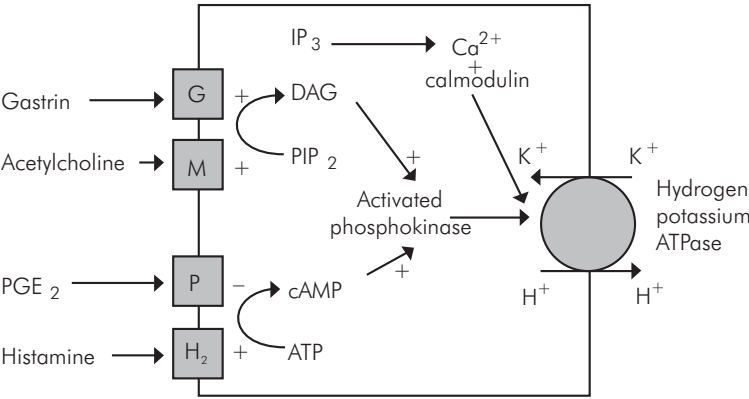


Figure 1.4.4.2 GI11 Modulators of gastric acid production.
 Reproduced with permission from Smith, T., Pinnock, C. and Lin, T. 2009. *Fundamentals of Anaesthesia*. Cambridge: Cambridge University Press. © Cambridge University Press 2009.

muscarinic receptors) and gastrin (via gastrin receptors). These receptors are located on the basolateral membrane of the parietal cell. The apical membrane contains an ATP-dependent hydrogen ion pump (Figure 1.4.4.1.).

The three receptors on the basolateral membrane utilise second messenger systems. Acetylcholine and gastrin increase intracellular concentrations of calcium via inositol triphosphate activation. The histamine receptors activate adenylyl cyclase via G-stimulatory proteins. This results in an increase in cyclic AMP levels. Increased calcium and cyclic AMP causes activation of specific protein kinases which increase the activity of the hydrogen ion pump.

Hydrogen ions are produced by the reaction of carbon dioxide with water under the influence of carbonic anhydrase. The hydrogen ions are then actively transported into the lumen by the hydrogen-potassium ATP-dependent pump. Bicarbonate ions pass

across the basolateral membrane in exchange for chloride ions via an antiport mechanism.

Physiological control of gastric acid production may be considered in terms of cephalic, gastric and intestinal factors.

Cephalic factors, such as anticipation, sight, smell and taste are mediated via the vagus nerve. This occurs before food ingestion and can be responsible for up to 50% of gastric acid production.

The gastric phase is initiated by the entry of food into the stomach. Acid secretion is stimulated in several ways. Distension of the stomach activates mechanoreceptors stimulating vagal reflexes, gastrin release from G cells and acid secretion from parietal cells. The acidic pH enhances pepsinogen secretion mediated by local reflexes.

The intestinal phase is initiated as chyme enters the duodenum stimulating further gastrin release, but this is of minor importance compared with cephalic and gastric factors.

What factors predispose to vomiting or regurgitation and aspiration of gastric contents?

Vomiting is an active process. As it is more likely to occur during the lighter planes of anaesthesia it is more common during induction of, or emergence from, anaesthesia. It should not occur during maintenance of anaesthesia.

Regurgitation is a passive process. Therefore, it may occur at any time and is not always immediately apparent. Because regurgitation usually occurs during deep anaesthesia, laryngeal reflexes are absent and the risk of aspiration is high.

Factors associated with regurgitation include:

- Non-elective surgical procedures
- Difficult airway or intubation
- Light plane of anaesthesia
- Gastrointestinal reflux
- Hiatus hernia
- Upper or lower gastrointestinal pathology
- Obesity
- Opioid medication
- Alcohol
- Lithotomy position
- Pregnancy.

A patient with a known hiatus hernia presents for an emergency laparotomy. What drugs would you use preoperatively to reduce the risk of regurgitation and aspiration?

My aims in this case would be to promote gastric emptying, reduce gastric acid production and neutralise gastric acidity.

To promote gastric emptying, I would give metoclopramide. Its prokinetic actions are mediated by antagonism of peripheral dopaminergic receptors and selective stimulation of gastric muscarinic receptors. This results in an increased lower oesophageal sphincter tone and relaxation of the pylorus.

To reduce gastric acid production, I could use either a histamine two (H₂) receptor antagonist such as ranitidine or a proton pump inhibitor such as omeprazole or lansoprazole. Ranitidine is a competitive antagonist of H₂ receptors at parietal cells. The gastric pH is raised and the volume of secretions is reduced. It does not affect the lower oesophageal sphincter. Omeprazole reversibly blocks the hydrogen ion pump in the apical membrane of the parietal cell, the final common pathway of gastric acid secretion. Again, it does not affect lower oesophageal sphincter tone. Proton pump inhibitors have a greater potential to achieve absolute inhibition of acid secretion compared with H₂ receptor antagonists.

I would use an antacid such as sodium citrate to raise the pH of gastric contents immediately before induction and 30 mmol of sodium citrate just prior to induction is another alternative.

1.4.5 Pancreatitis – Matt Thomas

This is presented as a short case scenario but could also be used as a long case with the history and examination findings provided along with blood results and radiology.

The surgical registrar asks you to see a previously well 61 year old man with a 2 day history of epigastric pain and vomiting. He has oxygen saturations of 92% on 5 l/min of oxygen, a heart rate of 115 bpm and a blood pressure of 85/50 mmHg after 2 litres of 0.9% sodium chloride. He is tender in the epigastrium and right upper quadrant.

What is the differential diagnosis?

The differential diagnosis of an acute abdomen with shock is wide. In this case I would consider particularly gastric, duodenal, biliary, and pancreatic pathology, with the possibility of more remote causes of peritonitis, aortic disease or even an MI in the back of my mind.

The lipase is 2200 U/L. What do you think the diagnosis is and how could you confirm this?

Pancreatitis may be diagnosed on the basis of the abdominal pain consistent with this disease and the lipase which is more than three times the upper limit of normal. Although other upper GI pathology can cause elevated lipase, at this level with this presentation pancreatitis is the most likely diagnosis.

Ultrasound or CT of the pancreas may show pancreatic swelling. Contrast enhanced CT is particularly good for confirmation as it can show pancreatic inflammation even if lipase is not elevated. These tests are also useful to determine the cause of pancreatitis (such as gallstones) or to exclude other pathology in uncertain cases. An ECG, cardiac enzymes and chest X-ray should be done in all cases for the same reason.

You've mentioned gallstones. What are the other common causes of pancreatitis?

Overall, gallstones account for about 50% of cases and alcohol for another 20–25%. Pancreatitis in women is more likely than this to be gallstone-related and in men it is more frequently due to alcohol. In up to 20% of cases no cause is found. Other less common causes include drugs, trauma, endoscopic retrograde

cholangiopancreatography (ERCP), infections; particularly viral and including HIV, hypertriglyceridaemia, hyperparathyroidism, malignancy and autoimmune diseases.

What is the underlying pathophysiology?

This remains controversial but is believed to be unregulated activation of trypsin within pancreatic acinar cells. Local cell damage and inflammation results, with activation of complement and kinin pathways and stimulation of neutrophils and macrophages. This results in leucocyte migration and release of further pro-inflammatory cytokines and reactive oxygen species leading to further acinar cell injury and necrosis. About 20% of cases are severe in which the local inflammation leads to a systemic inflammatory response and extra-pancreatic organ dysfunction, or there is a local complication such as pancreatic pseudocyst, pseudoaneurysm, or necrosis.

You have touched on severity. How do you define severity of acute pancreatitis?

The revised Atlanta Classification of mild, moderate and severe should be used. Mild pancreatitis has no associated organ failure or complications. Moderately severe pancreatitis is associated with transient organ failures resolving within 48 hours, or complications in the absence of organ failure. In severe acute pancreatitis organ failure persists beyond 48 hours.

Can you predict which attacks will be severe?

There are a number of scoring systems using a variety of clinical, laboratory and in some cases radiological factors, perhaps indicating that predicting the course of pancreatitis on admission is difficult. In the United Kingdom the Glasgow score is often used, where a score of 3 or more predicts a severe attack, although strictly speaking this requires 48 hours to complete. Before 48 hours prediction is harder, but a rise in urea in the first 24 hours of admission is a risk factor for mortality. Finally, the CT severity index, which may be calculated on the day of admission if a CT is done, shows good correlation with complications, sepsis, need for ICU care and mortality.

What are the components of the Glasgow score?

If you really want to remember these try using the mnemonic PANCREAS for pO₂, age, neutrophils (i.e., WCC), calcium, renal (urea), enzymes (LDH or ALT), albumin and sugar (glucose).

The Glasgow score uses eight parameters: pO₂, age, white cell count, calcium, urea, either ALT or LDH, albumin and glucose. Each may be scored as 0 or 1. I can't remember the cut-off values to score points, but if 3 or more points are scored within the first 48 hours the attack is a severe one. Practically speaking the number of failing organs, and the degree of failure, is a good indicator of the severity of the attack and is much more useful information for the intensivist.

This man's score predicts severe acute pancreatitis, and ultrasound shows a stone in the common bile duct. Will you admit him to the HDU?

Yes, I would admit for three reasons. Firstly, whatever the score predicts, he currently is seriously unwell with evidence of organ failure in that he is hypotensive despite

some fluid resuscitation and is hypoxic. Secondly, he has been unwell for 48 hours and is likely to get worse over the next 48 hours, especially as there is a difficult balance between fluid required for haemodynamic and renal support and his precarious respiratory function. Finally, patients with acute severe pancreatitis need a lot of medical and nursing input that frequently is too much for a ward area. This is true even if no formal organ support, such as vasopressors or renal replacement, is currently needed although in fact you could consider high volume fluid resuscitation as a form of cardiovascular support.

How will you manage him once in your HDU?

Management of severe pancreatitis is aimed at the support of organ function and prevention of complications, as there is no therapy for pancreatic inflammation once established. Adequate early – that is within 24 hours of onset – resuscitation with oxygen and fluid is probably the most effective way of reducing the risk of later organ failure. However, as many patients reach the HDU or ICU days after the onset of disease there is a risk fluid resuscitation will not reverse and may exacerbate organ failures. I would titrate resuscitation to dynamic measures like reduction in serum lactate or improvement in peripheral capillary refill rather than a fixed volume of fluid or a CVP. Vasopressors are often required to maintain pressure and perfusion but there is no consensus on early or late use.

Yes, early intervention is important. Is there anything else?

Analgesia is important, and this could be provided with either strong opioids or even an epidural if the benefits were thought to outweigh the risks. Nutrition is vital as this will reduce the incidence of infectious complications, and I would begin enteral feeding as soon as haemodynamic stability was achieved, with the aim of meeting requirements as soon as possible. DVT prophylaxis should be used in the absence of contraindications. Once stable, the cause of the pancreatitis should be addressed. If the cause of pancreatitis is gallstones then ERCP may be indicated.

There's a lot there to talk about. I'll take your last point first.

How soon should ERCP be done? Is it an easy procedure?

If there is cholangitis or bile duct obstruction associated with the gallstones this should ideally be done within 48 hours. This will allow sphincterotomy with or without stenting to relieve obstruction. It requires screening facilities and can be difficult to tolerate and to perform, so in most critically ill patients elective intubation and ventilation will be required. An alternative is percutaneous transhepatic cholecystotomy (PTC) – drainage of the obstructed biliary system that can be done under local anaesthesia.

Why go for a general anaesthetic when most ERCPs are done with sedation?

Patients in the HDU with severe acute pancreatitis have compromised respiratory function, large distended painful abdomens with a significant risk of a full stomach and are often confused. Sedation would carry risks of loss of the airway, aspiration, further hypoxia and worsening confusion.

What about cholecystectomy?

This should wait until organ failure has resolved, but ideally will be done during the same admission to prevent recurrent pancreatitis.

Nutrition has been a contentious issue. Is enteral nutrition really safe?

It is true that older strategies emphasised 'resting the gut' and avoiding enteral nutrition which was thought to increase pancreatic inflammation. However, recently the advantages of enteral nutrition have become clearer, in particular maintenance of gut barrier function, a reduction in septic episodes, fewer surgical interventions and fewer non-infectious complications, and a difference in mortality in meta-analysis. It is also cheaper than TPN, especially when a shorter hospital stay is taken into account.

Most evidence is from trials of nasojejunal feeding, although direct comparisons of NG and NJ feeding suggest they are equivalent and NG feeding is possible in up to 80% of cases. However, gastric outflow obstruction and ileus are common in severe acute pancreatitis so enteral feeding may be difficult to establish and underfeeding is a serious risk, as is regurgitation, vomiting and aspiration. If adequate NG or NJ feeding cannot be established within 3 to 5 days then parenteral supplementation or TPN should be used as calories are ultimately more important than the route.

Practically speaking, how would you attempt to establish feeding?

I would start using the NG route, and use prokinetics early and in combination if aspirates are high in the first 24 hours. If there is no improvement in the next 24 hours I would move from NG to NJ feeding. If this is poorly tolerated, for example if there is abdominal pain, distension, reflux, nausea or vomiting, then I would use parenteral nutrition. It's important to agree this between all those likely to be involved in the patient's care, especially surgical colleagues.

What about supplements?

Pancreatic enzyme supplements are now routinely prescribed alongside oral and enteral feeds to mitigate any exocrine deficiency associated with acute pancreatitis. I would take advice from a dietitian on the appropriate enzyme supplement and whether any other trace elements or vitamins are needed.

Well, we could discuss your choice of resuscitation fluid, or your use of surviving sepsis targets, or even whether an epidural would ever be indicated in this situation. However, time is pressing. So tell me, what are the complications of pancreatitis?

These may be divided into local and systemic complications. Systemic complications arise earlier and are essentially extra-pancreatic organ failures; these are responsible for most early deaths. ARDS, acute kidney injury and gut failure and the abdominal compartment syndrome are most common and, as with other causes of the systemic inflammatory response, patients are more susceptible to infections. Of local complications, pancreatic necrosis may also develop early. Other local complications may take a

week or two to develop and include pseudocysts, pseudoaneurysm, venous thrombosis and pancreatic necrosis. There may be a late failure to improve or subsequent deterioration if necrosis becomes infected. There may also be therapy-related complications particularly from ERCP.

Tell me more about pancreatic necrosis.

The extent of pancreatic necrosis seen on CT is a guide to the severity of the condition. However, what is really significant is the fact that the leading cause of morbidity and mortality is infected pancreatic necrosis. Somewhere over 50% of patients with severe pancreatitis will develop infected necrosis by the second or third week of their illness, so there has been a lot of interest in preventing infection which leads to the question of antibiotic prophylaxis.

Ah, yes, I wondered when you would mention that. Antibiotics are often used. Should they be?

Yes, broad-spectrum antibiotics are often started in severe acute pancreatitis with the intention of reducing the incidence of infected necrosis and mortality. NCEPOD suggested 20% of courses were inappropriate. There is conflicting evidence but more recent and higher quality trials are associated with reduced or no treatment effect so current guidelines do not recommend prophylactic antibiotics.

What about probiotics or selective decontamination of the digestive tract?

Both interventions are concerned with manipulating the gut microbiome, which is a key component of critical illness generally and pancreatitis specifically. Understanding the mechanisms and impact of targeted therapy is at an early stage, however, and evidence for these interventions is inconclusive. It is an area where things are likely to develop but at present routine use is not recommended.

So what will you do for this man? Will you start antibiotics? Let us say it is day 7 and he is now ventilated and on noradrenaline, with a creatinine of 350 $\mu\text{mol/L}$ and rising, despite successful ERCP on day 3. CT shows about one third of his pancreas is necrotic.

Infection is unlikely if less than 30% of the pancreas is necrotic. This man has evidence of progressive multiorgan failure. In this situation I would start empirical antibiotics after full septic screen perhaps including a CT-guided fine needle aspiration (FNA) of the necrosis. I'd be guided by microbiological advice, but meropenem would be an appropriate choice for both spectrum of activity and penetration of pancreatic tissue, and I would plan a course no longer than 14 days. Anti-fungal cover is usually not necessary unless there have been previous antibiotics used or other risk factors such as TPN use or surgical intervention. A baseline beta-D-glucan might be helpful. At this stage with evidence of necrosis and a deteriorating course I would discuss the case with the specialist pancreatic centre within the referral network.

What is the role of CT scanning?

As said before, the CT severity index is a good prognostic guide early in the course of the disease, but unless there are other diagnostic indications for a CT it should not be done purely for staging purposes. Necrosis may develop throughout the first week, so early CT may underestimate the final extent and severity. A CT should be done if there are new or persisting symptoms, signs, sepsis or organ failures as local complications may have occurred and require intervention.

So when is surgery indicated?

This is a question best decided in consultation with surgical and radiological colleagues following the principles of ‘delay, drain, debride’. Cases of infected necrosis may not resolve without necrosectomy and waiting does not increase mortality and may reduce the number of interventions. A step-up approach is used: endoscopic and percutaneous drainage, then minimal access or video-assisted debridement or laparoscopic necrosectomy. Some patients need an open necrosectomy, repeat laparotomies or continuous drainage of the pancreatic bed.

Thank you, we’ll finish there.

Further Reading

Banks P, Bollen T, Dervenis C, et al.

Classification of acute pancreatitis – 2012: Revision of the Atlanta classification and definitions by international consensus. *Gut*. 2013; 62: 102–111.

Crockett S, Wani S, Gardner T, Falck-Ytter Y, Barkun A. American Gastroenterological Association Institute Guideline on Initial Management of Acute Pancreatitis. *Gastroenterology*. 2018; 154: 1096–1101.

Gomes C, Di Saverio S, Sartelli M et al. Severe acute pancreatitis: Eight fundamental steps revised according to the ‘PANCREAS’ acronym. *Annals of the Royal College*

of Surgeons of England. 2020; 102: 555–559.

National Confidential Enquiry into Patient Outcome and Death. Acute pancreatitis: Treat the cause. 2016. www.ncepod.org.uk/2016ap.html accessed 22 August 2022.

National Institute for Health and Care Excellence National Guideline 104: Pancreatitis. www.nice.org.uk/guidance/ng104 accessed 22 August 2022.

Trikudanathan G, Wolbrink D, van Santvoort H, Mallory S, Freeman M, Besselink M. Current concepts in severe acute and necrotizing pancreatitis: An evidence-based approach. *Gastroenterology*. 2019; 156: 1994–2007.

Haematological

1.5.1 Blood Groups – Sophia Henderson and Rebecca A Leslie

What is a blood group system?

Blood groups result from different antigens expressed on the surface of red blood cells (RBCs). There are a number of different blood group antigens, and those produced by the same gene are called a blood group system. Two of the most important blood group systems are the ABO system and the Rhesus (Rh) system as these are the main systems responsible for transfusion compatibility. However, these are only 2 of 29 blood group systems which have currently been identified. Examples of other systems are the Kell, Kidd, Lutheran and Duffy systems. The importance of these other blood groups systems should not be underestimated.

Tell me more about the antigens and antibodies in the ABO blood group system.

The ABO system describes four different blood groups:

- Blood group A which has A antigens (agglutinogens) on their RBCs
- Blood group B which has B antigens on their RBCs
- Blood group AB which has both A and B antigens on their RBCs
- Blood group O which has no antigens on their RBCs.

Figure 1.5.1 shows the relative frequencies of the ABO groups in the United Kingdom.

Early in life individuals develop antibodies (called agglutinins) in the plasma against non-self antigens. These antibodies do not require exposure to different types of blood, but instead develop because antigens similar to A and B antigens are found in the gut and in some foods which individuals are exposed to. This is thought to occur early in neonatal life. As a result, anti-A antibody is present when the A antigen is absent and anti-B antibody is present when the B antigen is absent. Therefore, in summary, individuals who have blood group A with A antigens expressed on their RBC will have anti-B antibodies within their blood. Similarly, individuals with blood group O will have anti-A and anti-B antibodies, and those with blood group AB will have no antibodies.

This explains why patients with blood group AB are the ideal recipients, as they have no antibodies that will agglutinate ('clump') the donor blood. Equally blood group O is the ideal donor blood because once the antibodies are removed from the plasma, there are no antigens expressed on the RBC surface to agglutinate with antibodies in the recipient's blood.

Blood group	Naturally occurring antibodies (IgM)	UK (%)
O	Anti-A, anti-B	47
A	Anti-B	42
B	Anti-A	8
AB	None	3

Figure 1.5.1 Relative frequencies of ABO groups.

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Can you tell me about the inheritance of A and B antigens?

A blood group is encoded on a single pair of genes, with each gene being one of three alleles: A, B or O. These alleles are inherited by Mendelian dominance. Therefore, a patient with blood group A could have the genotype AA or AO, whilst a patient with blood group B, could have the genotype BB or BO. However, an individual with blood group O must have the genotype OO, and blood group AB the genotype AB.

Tell me more about the Rhesus blood group.

The Rhesus blood group system is the second most important after the ABO blood group system. It is very complex and comprises many different antigens, including C, D and E antigens. D antigen is the most important antigen as it is felt to be the most antigenic. When a patient is described as being 'Rhesus positive', this term normally is being used to describe the presence of D antigen on the individual's RBCs.

In contrast to the ABO blood group system, anti-D antibodies are not normally found in the blood of Rhesus D-negative individuals. Instead, anti-D antibodies only develop if the individual's blood comes into contact with Rhesus D-positive blood. This can occur by either an inappropriate blood transfusion or by the entry of fetal Rhesus D positive blood into the circulation of a Rhesus D negative mother. These anti-D antibodies will cause potential problems if the individual subsequently receives transfusion of further Rhesus D positive RBCs.

What causes haemolytic disease of the newborn?

If a Rhesus D negative mother gives birth to a Rhesus D positive baby, as explained above, it is likely she will develop anti-D antibodies due to exposure to a small amount of the fetal RBCs (normally occurring at birth). The anti-D antibody is an immunoglobulin G (IgG) of small molecular mass (150 kDa) and is able to cross the placenta and enter the fetal circulation. This means that in future pregnancies the anti-D antibodies could cross the placenta and cause haemolytic disease of the newborn in Rhesus D positive babies.

How do we prevent haemolytic disease of the newborn?

In the postpartum period, we administer a single dose of Rhesus immunoglobulin to prevent sensitisation of the mother. This practice has reduced the incidence of haemolytic disease of the newborn by 90%.

How is a 'group and save' performed?

Initially the patient's ABO and Rhesus blood group is determined by combining the patient's blood with monoclonal typing reagents; anti-A, anti-B, anti-A+B and anti-D.

Next, to ensure the correct ABO blood group has been identified, a reverse group is performed. This is where the patient's serum is mixed with both the group A and group B red blood cells. For example, if the patient's blood is type A, when it is first combined with anti-A antibodies agglutination ('clumping') will occur; however, there will be no agglutination when it is combined with anti-B antibodies. Next to ensure that it definitely is blood group A, a sample is combined with blood known to be group A to ensure there is no agglutination, and a sample is combined with blood known to be group B, to demonstrate agglutination.

Next the blood is screened for the presence of antibodies. The sample is combined with a sample of red blood cells which are known to carry all common red blood cell antigens. If the test is positive, then the antibody is identified by testing the patient's serum against a panel of individual RBCs.

How is a cross-match performed?

After a 'group and save' has been performed, samples from donor blood are combined with samples of the patient's blood to ensure no reaction occurs. This is particularly important to detect antibody/antigen interactions which may occur due to other, less common blood group systems.

When would you consider giving your patient a blood transfusion?

The purpose of giving a blood transfusion is to improve the oxygen-carrying capacity of the blood. A blood transfusion is not without risk, and each patient should be assessed, on an individual basis, prior to considering a blood transfusion.

In accordance with the recent AoA guidelines I would tend not to transfuse anyone who has a haemoglobin >70 g/L; with a target of 70–80 g/L post-transfusion. However, it is important to recognise that there are situations where a blood transfusion in these patients might be appropriate.

It has been suggested that even in the elderly and those with cardiorespiratory disease a haemoglobin >80 g/L is sufficient. However, if their haemoglobin were below this level or if they were symptomatic, they should be transfused.

How would you give a blood transfusion?

The British Committee for Standards in Haematology (BCSH) has issued guidelines on the administration of blood transfusions. The AoA have also published a guideline for blood transfusion and the anaesthetist. I follow both these guidelines in my clinical practice. Outlined below are the steps for a manual checking method, although the preferred method would be an electronic transfusion management system.

- Confirm identity of patient (all patients receiving blood must be wearing an identification name band). Minimum patient identifiers are first name, last name, date of birth (DOB) and unique hospital number.
- Immediately before the transfusion, check the component next to the patient, against the prescription.
- Check the blood compatibility label to ensure the blood is the correct type for the patient. Inspect the bag to ensure integrity of plastic casing.

- Administer the blood using a blood administration set with an integral mesh filter (170–200 microns).
- Transfusion should be completed within 4 hours of leaving the blood fridge.
- Details of the blood transfused must be recorded on the anaesthetic chart or in the medical notes.
- The patient should be monitored regularly during the blood transfusion.

How can the need for an allogenic blood transfusion be minimised in surgical patients?

Remember to classify your answer.

The need for a blood transfusion can be reduced by preoperative and intraoperative measures.

Preoperatively, appropriate assessment is key to minimising the need for a blood transfusion:

- Elective surgery in anaemic patients should be postponed until they have been appropriately investigated and treated
- Patients on anticoagulants and/or antiplatelet agents should have their medications stopped for the pre-requisite time period prior to surgery
- Early haematological advice should be sought in patients with bleeding and coagulation disorders
- Consider minimally invasive or laparoscopic surgical techniques
- Point-of-care testing should be available with appropriate training.

Intraoperative blood loss can be minimised by:

- Use of tourniquets
- Careful patient positioning to maintain venous drainage
- Maintenance of body temperature $> 36^{\circ}\text{C}$
- Usage of cell salvage if intraoperative blood loss > 500 ml
- Local infiltration of vasoconstrictors
- Regional anaesthesia
- Using a hypotensive anaesthetic technique
- Pharmacological therapy; tranexamic acid if blood loss > 500 ml and desmopressin (in patients with von Willebrand disease or mild haemophilia A).

What are the risks of blood transfusion?

The risks associated with a blood transfusion can be classified as immunological, infectious and physiological risks.

Immunological:

- Immediate haemolytic transfusion reaction. This occurs due to ABO incompatibility and is normally due to a human error. Antibodies in the recipient's blood interact with antigens on the donor blood causing agglutination of donor cells. This leads to microvascular blockage and haemolysis.

- Delayed haemolytic transfusion reaction. These reactions occur when a patient has developed a red cell antibody due to a previous pregnancy or transfusion and the low titre of this antibody escaped detection in the 'group and save'. These reactions can present up to several days after the transfusion and symptoms and signs include fever, jaundice, fatigue, a drop in haemoglobin and haemoglobinuria.
- Febrile non-haemolytic transfusion reaction. This is the most common type of transfusion reaction. It occurs as a result of recipient anti-leucocyte antibodies. These reactions present with an increased temperature, and – when severe – nausea, vomiting, rigors and collapse.

Anaphylaxis:

- Urticarial reactions. These occur due to plasma proteins in the donor blood.
- Transfusion-related acute lung injury (TRALI). This is defined as an acute lung injury which occurs within 6 hours of transfusion in the presence of no other risk factors for ARDS. It is most common after FFP transfusion. It is caused by anti-leucocyte antibodies in the donor blood. Unlike ARDS, it tends to resolve within 48 hours of the transfusion.

Infectious:

- Hepatitis B, hepatitis C, HIV, HTLV, malaria, cytomegalovirus, brucellosis, bacterial contamination and prion diseases can all be transmitted through contaminated blood; however, in the UK the risk is small.

Physiological:

- Electrolyte disturbance: hyperkalaemia, hypocalcaemia and acidosis
- Transfusion-associated circulatory overload can occur in susceptible patients
- Dilutional coagulopathy
- Dilutional thrombocytopenia
- Hypothermia.

When would you consider using intraoperative cell salvage?

I would consider use of intraoperative cell salvage:

- Anticipated blood loss of >500 ml
- Patients with a rare blood group or multiple antibodies
- Patients with low haemoglobin or increased risk of bleeding
- Patient objections to the use of allogenic blood (Jehovah's Witnesses).

Further Reading

Association of Anaesthetists of Great Britain and Ireland (AoA) Guidelines: The use of blood components and their alternatives. *Anaesthesia*. 2016; 71(7):829–842.

British Committee for Standards in Haematology (BCSH). Guideline on the Administration of Blood Components. First published 2009. [www.bcsguidelines](http://www.bcsguidelines.com/4HAEMATOLOGYGUIDELINES.html?type=Transfusion&dpage=0&sspage=0&ipage=0)

[.com/4HAEMATOLOGYGUIDELINES.html?type=Transfusion&dpage=0&sspage=0&ipage=0](http://www.bcsguidelines.com/4HAEMATOLOGYGUIDELINES.html?type=Transfusion&dpage=0&sspage=0&ipage=0).

Kirkman E. Blood groups. *Anaesthesia and Intensive Care*. 2010; 11(6): 232–235.

Mugabe B et al. Serious hazards of transfusion (SHOT): Its implications for intensive care *Journal of the Intensive Care Society*. 2013; 14(3): 215–219.

1.5.2 Anaemia and Abnormal Haemoglobins – Farzad Saadat and Sarah F Bell

Haematology is a topic that comes up again and again in the final exam since it is vital that we have a good understanding of key topics such as anaemia. Questions could come up anywhere in the exam so be prepared!

You are reviewing a 77-year-old woman with rheumatoid arthritis for a revision of her total hip replacement. She has no other medical conditions. Her blood results reveal a haemoglobin concentration of 90 g/L with a mean corpuscular volume of 80 femtolitres/cell and a mean corpuscular haemoglobin of 30 picograms/cell. The white cell count is $6 \times 10^9/\text{L}$ and her platelet count is $450 \times 10^9/\text{L}$.

What can you tell me about the haematology results?

This woman is anaemic. The normal haemoglobin concentration for a woman is 110–150 g/L and 130–160 g/L for men. The MCV and MCH are within normal limits, indicating that the anaemia is normochromic and normocytic.

What is the difference between the MCV and MCH?

The mean cell volume, or MCV, is a measure of the average red blood cell volume. The normal reference range is typically 80–100 femtolitres/cell. In contrast, the MCH is the average mass of haemoglobin per red blood cell in a sample of blood. A normal value is 27–31 picograms/cell.

What are the clinical features of anaemia?

The features will, to a certain extent, depend on the speed at which the anaemia has developed. If the red blood cell concentration has fallen slowly, the patient may compensate and be asymptomatic. Nonspecific symptoms such as fatigue, headache and syncope may occur; as may dyspnoea, angina, intermittent claudication and palpitations. These features will be more pronounced in an acute situation and the patient may become confused.

On examination the patient may again have nonspecific signs such as pallor, tachycardia, flow murmurs and ankle oedema due to cardiac failure. More specific signs may further define the cause of the anaemia. For example, koilonychia may be found in iron deficiency anaemia, jaundice might indicate haemolysis, bone deformities may be found in thalassaemia and leg ulcers in sickle cell disease.

What are the causes of anaemia?

This question can be answered in a number of different ways depending on how you want to classify the causes. This is one way. Describing the type of anaemia is another (i.e. normocytic or macrocytic).

I would split the causes into reduced production of red blood cells, increased breakdown (haemolysis) and blood loss, which might be acute or chronic.

There are many potential causes of reduced production of red blood cells. Deficiencies of key components of red blood cells such as iron, vitamin B12 or folate will cause reduced production; as may chronic diseases (such as malignancy or infection) or endocrine conditions such as hypothyroidism or adrenocortical insufficiency. Bone

marrow infiltration due to leukaemia or myelofibrosis may also cause reduced production, as may aplastic anaemia. Furthermore reduced erythropoietin secretion, alcoholism, liver and renal disease may all decrease production of red blood cells.

Increased red blood cell breakdown can be subdivided into conditions that are inherited or acquired.

Inherited diseases may affect the red cell membrane leading to increased sequestration in the spleen. This may occur in hereditary spherocytosis. The abnormal haemoglobin containing cells observed in patients with thalassaemia or sickle cell disease are less deformable and will also become sequestered in the spleen. Finally metabolic defects such as glucose 6 phosphate dehydrogenase deficiency will also cause inherited haemolytic anaemia (due to altered cell membrane characteristics triggered by oxidative stress).

Acquired red blood cell breakdown can be classified into autoimmune, iso-immune, and non-immune conditions.

In autoimmune conditions the body's immune system attacks its own red blood cells. Warm and cold haemolytic anaemias may occur. In the cold condition the antibodies only bind to red blood cells at low body temperatures of about 28–31 degrees centigrade. The test for the presence of antibodies against red blood cells is the direct Coombs test. Certain drugs can also stimulate the productions of antibodies against red blood cells. Isoimmune conditions include Rhesus or ABO incompatibility. Non-immune causes of haemolytic anaemia include red cell membrane defects such as paroxysmal nocturnal haemoglobin and diseases affecting the liver and kidneys. Mechanical trauma or destruction of red blood cells may occur in patients with microangiopathic haemolytic anaemia, valve prostheses, march haemoglobinuria or sepsis.

What blood tests would you consider for investigating anaemia?

The blood tests would depend on the type of anaemia present.

If the patient presented with a hypochromic, microcytic anaemia then causes such as iron deficiency, anaemia of chronic disease or thalassaemia should be considered. Appropriate initial investigations would include assessment of iron status. In iron deficiency anaemia a low serum ferritin, low serum iron and high total iron binding capacity would be expected. Haemoglobin electrophoresis would identify thalassaemia.

A macrocytic anaemia should be investigated by assessment of vitamin B12 and folate levels. If abnormal, testing for parietal cell and intrinsic factor antibodies will aid diagnosis of pernicious anaemia. Furthermore, tests such as thyroid function, liver function and a pregnancy test should also be considered.

A normochromic, normocytic anaemia might be due to acute blood loss, anaemia of chronic disease, aplastic anaemia, combined deficiency, haemolytic anaemias or endocrine disorders. Investigations would therefore include B12 and folate levels, iron studies, evidence of haemolysis such as elevated serum bilirubin and lactate dehydrogenase. The blood film should be assessed for sickle cells, parasites and reticulocytes. The platelet count and white blood cell count and morphology might also be investigated. Endocrine tests such as thyroid, pituitary and adrenal function might be appropriate in some cases.

What are the potential physiological effects of anaemia?

There are a number of effects of anaemia. Firstly there will be a reduced oxygen-carrying capacity leading to fatigue, dyspnoea and angina. Secondly, the cardiac output increases to

maintain oxygen flux which can cause palpitations and tachycardia. Thirdly, anaemia leads to an increase in 2,3 DPG and a shift of the oxyhaemoglobin dissociation curve to the right. There will also be the effects of the disease or condition causing the anaemia.

What can you tell me about vitamin B12 deficiency?

B12 deficiency causes a macrocytic anaemia due to problems in DNA synthesis (specifically thymine). It has a number of causes including inadequate dietary intake, impaired absorption, chronic fish tapeworm infestation and drugs such as metformin that interfere with absorption. The impaired absorption may be due to intrinsic factor deficiency or surgical resection of the terminal ileum. Treatment requires identification of the cause of the deficiency and then replacement.

And what about folic acid deficiency?

A deficiency of folate will occur if the body's folate requirements increase, dietary intake is inadequate, or when the body excretes more folate than usual. Circumstances in which folate requirements increase include pregnancy, lactation, malabsorption syndromes and renal and liver disease. Many drugs interfere with folate utilisation including anticonvulsants, metformin, sulfasalazine and methotrexate.

What are the causes of iron deficiency anaemia?

Iron deficiency may be due to chronic bleeding, inadequate intake, malabsorption or drugs interfering with iron absorption. Gastric acidity enhances solubility and therefore the availability of iron derived from food. Achlorhydria or alkaline drugs such as antacids can therefore interfere with iron absorption, as can medications such as H2 receptor antagonists.

You are the registrar on call for emergency theatres and are called to see a patient with acute appendicitis. She is a 24-year-old Afro-Caribbean woman who tells you that her only medical condition is sickle cell disease.

What can you tell me about this condition?

Sickle cell is caused by an alteration in the amino acid sequence for haemoglobin. Specifically, it is due to the substitution of glutamic acid by valine in the sixth amino acid of the beta chains. The condition has an autosomal inheritance. Heterozygotes are described as having sickle cell trait and possess both normal and abnormal chains, whilst homozygotes contain only abnormal genes and are said to have sickle cell disease.

Sickle cell trait confers relative resistance to malaria and is therefore thought to have propagated through certain at-risk populations.

The condition is most prevalent in African and Asian populations. There are 15,000 affected individuals in the UK and 100–200 affected pregnancies each year.

How do you diagnose sickle cell disease or trait?

The diagnosis is made by taking a full history (including family history) and examining the patient. The sickledex test is a quick laboratory test but this will not differentiate between disease and trait. The gold standard test is electrophoresis which will provide the clinician with a definite diagnosis.

What are the differences between sickle cells and normal red blood cells?

The haemoglobin in a sickle cell has an abnormal β polypeptide chain, causing it to polymerise and precipitate when deoxygenated. This leads to formation of the sickle shape, which is more rigid than the normal flexible red blood cell. The sickle cell thus increases blood viscosity and thrombosis with a reduction in flow. The sickle cell also has a reduced lifespan. The partial pressure at which the haemoglobin in sickle cells polymerises depends on whether the patient has disease or trait. In patients with the disease this may occur at partial pressures below 6 kPa (and so can happen continuously), whilst in trait the haemoglobin will sickle at lower partial pressures of 2.5 to 4 kPa. Oxygen affinity is normal when the cell is not polymerised.

What features of sickle cell disease would you look for in your preoperative assessment of this patient?

The features can be divided into those associated with haemolysis, impaired blood flow and end organ damage.

The haemolysis associated with sickle cell typically causes a normochromic normocytic anaemia. The symptoms and signs of anaemia may be present, depending on its severity. There may be an associated hyperbilirubinaemia and jaundice. Patients may also develop gallstones. Furthermore bone marrow hyperplasia can lead to skull and long bone enlargement. I would want to know the patient's transfusion history and baseline Hb.

The altered blood flow has a number of effects on the different organ systems. There can be chronic changes and acute crises precipitated by conditions such as hypothermia, dehydration, infection, exertion and hypoxaemia.

With regard to the neurological system the patient may have a history of stroke or present with an acute neurological lesion. The respiratory system may be affected by pulmonary infarcts, pulmonary hypertension and right ventricular failure. Acute chest syndrome may occur with symptoms of pleuritic chest pain, fever, tachypnoea and pulmonary infarcts. The kidneys may be affected by infarction and the development of papillary necrosis and renal impairment. The musculoskeletal system may again suffer infarction and avascular necrosis. The gastrointestinal system may be affected by bowel ischaemia and splenic infarction or hyposplenism. These patients often require vaccination against pneumococcus, haemophilus and meningococcus and may require prophylactic antibiotics. The cardiovascular system may be affected by high output cardiac failure due to anaemia and increased risk of thrombotic events. Finally the patient may have ulcers due to impaired blood flow, retinopathy due to arterial occlusion and priapism can occur.

Additional features of sickle cell anaemia include an increased susceptibility to infections such as salmonella osteomyelitis and delayed growth and development in children.

What would you look for if the patient only had sickle cell trait?

The patient may be asymptomatic, but I would still look for all of the features of sickle cell disease because trait in combination with other haemoglobinopathies can produce more rigid cells that may sickle at a higher partial pressure of oxygen (for example HbCS).

The surgeons are keen to proceed with the case. Are there any preoperative measures that you would instigate before taking the patient to theatre?

I would want to take a full history and examine the patient. I would then review the full blood count, U + E's, liver function tests and ECG before going to theatre. I would check that blood was available for the patient if required. (In severe cases an exchange transfusion might be performed preoperatively to reduce the haemoglobin S concentration to below 40%, but this would depend on the urgency of the case and the patient's condition.) I would want to avoid precipitation of a sickle cell crisis and so would aim to prevent hypoxaemia, dehydration, hypothermia, acidosis and pain.

What would be your concerns intraoperatively?

Again, I would aim to prevent hypoxaemia, dehydration, hypothermia, acidosis and pain. In general I would also avoid using a tourniquet, although this would not be required in an appendicectomy!

What can you tell me about thalassaemia?

This disease is due to a lack of expression of a globin chain in the haemoglobin due to deletion or mutation. This leads to reduced production of alpha or beta chains. The severity of the condition is related to the pattern of inheritance of haemoglobin since one beta and two alpha genes are inherited from each parent.

The condition is more common in Mediterranean, African and Asian populations.

In beta thalassaemia the condition may not be immediately identified as fetal haemoglobin does not contain beta chains. Heterozygotes will have a mild anaemia whilst homozygotes develop a severe anaemia with craniofacial bone hyperplasia, hepato-splenomegaly and cardiac failure. Haemosiderosis will develop if the condition is treated with repeated blood transfusions.

Alpha thalassaemia patients are usually anaemic, the severity of which depends on the number of gene deletions.

Let's talk about anaemia during critical illness. Have you heard of the TRICC trial and what were the important findings?

The TRICC trial is over 20 years old, but still informs the transfusion guidelines of NICE, JPAC and others.

The TRICC trial stands for Transfusion Requirements in Critical Care. It compared a transfusion trigger of 70 g/L (with a target haemoglobin of 70–90 g/L), with a trigger of <100 g/L (and target of 100–120 g/L) in patients whose haemoglobin concentration was 90 g/L during the first 3 days of an ICU stay. Interestingly, the 30 and 60-day mortality was similar for both groups. Furthermore there was a significantly lower mortality with the restrictive strategy among younger patients (below 55) and fewer ill patients (APACHE score <20). This has led to the use of lower transfusion triggers in intensive care units.

Why do critically ill patients become anaemic?

Critically ill patients are often anaemic. There are many possible reasons for this. Haemodilution due to crystalloid or colloid based fluid resuscitation and/or blood loss

due to occult haemorrhage or frequent blood sampling often contributes. Additional causes include: reduced red cell survival due to the systemic inflammatory response syndrome causing cell destruction or altered membrane characteristics; or reduced red blood cell production due to bone marrow suppression. Alterations in B12 and folate metabolism, inappropriately low circulating erythropoietin concentrations and abnormal red blood cell maturation may all be caused by inflammatory cytokines interrupting normal pathways and thus lead to anaemia.

How can we manage anaemic patients on the intensive care?

There are several measures that can be incorporated into managing anaemia. These include attempting to reduce red cell loss by restricting blood sampling, using a closed blood sampling system and controlling haemorrhage; using appropriate transfusion triggers and recognising and treating any deficiencies promptly.

What would you consider to be exceptions to the restrictive strategy now employed?

Patients with chronic or acute ischemic heart disease may require a transfusion trigger of less than 80 g/L (rather than 70).

Further Reading

Herbert P, Wells G, Blajchman M et al. A Multicenter, Randomized, Controlled Clinical Trial of Transfusion Requirements in Critical Care. *New England Journal of Medicine*. 1999; 340: 409–417.

NICE blood transfusion guidelines. 2015.

Stoddard K, Sohal M, Bedson R. Anaesthetic management of patients with sickle cell disease in obstetrics. *British Journal of Anaesthesia Education*. 2022; 22(3): P87–93.

1.5.3 Abnormalities of Coagulation and Haemostasis including Massive Haemorrhage – Philip Harrington and Justin C Mandeville

Disorders of coagulation include those at risk of excessive bleeding and those at risk of excessive thrombosis.

A 23-year-old man needs an urgent laparotomy for a ruptured spleen. The team are happy that there are no other injuries, but he has mild haemophilia.

The patient is tachycardic but otherwise stable, and has large bore intravenous access and a bag of crystalloid is being infused.

You have time to take a brief history. What questions might you ask him?

I would take a brief general history and specifically ask about his haemophilia. I would ask particularly about previous episodes of bleeding, previous surgical procedures, any regular treatment he receives and any recent blood tests he might have had.

I would also like to be sure which factor was deficient. In haemophilia A this would be factor VIII and haemophilia B it is factor IX. If, for example he has haemophilia A, it would be useful to know a recent factor VIII level – a level of 50% or more would be

reassuring, but may still require replacement in active haemorrhage. Electively these patients should have their levels corrected to 80–100% but there is clearly not the time to be able to do this in this case. I would therefore seek the advice of the haematologist on call with regard to factor replacement. I would also ask the patient about previous blood transfusions as this may impact upon the time taken to perform blood cross-match.

What might a full blood count and coagulation studies show in this patient at this point?

As the blood loss is acute and there may not have been much fluid given yet it is possible that there will be little fall in the haemoglobin or haematocrit, though the platelets may fall through consumption. In haemophilia A the activated partial thromboplastin time is likely to be raised but the bleeding time and prothrombin would be normal.

The surgeons are keen to start straight away. Would you be happy to do that?

This is a balance between how quickly you can optimise the patient and make arrangements to safely proceed and the risks to the patient of delaying his surgery.

The patient is exhibiting signs of hypovolaemia, and although the blood pressure is stable, a fall in blood pressure is often a late sign of hypovolaemia in a young person. The tachycardia alone suggests he has lost at least a 15% of his circulating volume as a result of acute blood loss. Therefore I would like to assess the patient myself regarding their volume status, and ensure adequate fluid resuscitation prior to induction of anaesthesia.

I would ask for senior support with this case and I would also contact the haematologist for advice regarding specific blood products. I would request that 6 units of blood be cross-matched immediately, ready to give in theatre. Along with this I would ask for an adult dose i.e. 4 bags of fresh frozen plasma (FFP) to be thawed by the blood bank. As well as intravenous access, I would establish invasive arterial blood pressure monitoring prior to induction and ideally, central venous access.

Can you tell me about the blood products you mentioned that might be of use in the perioperative period?

Consider those specific to the haematological problem and those you might use in any major haemorrhage.

I would have packed red cells available if there were signs or evidence that blood loss was in excess of around 2 litres.

In an elective case factor VIII levels should be normalised for at least a week before major surgery. In emergencies such as this, factor VIII concentrate should be given as soon as possible. Haematologists should be closely involved, as a proportion of haemophiliacs have been found to have antibodies to factor VIII. Both fresh frozen plasma and cryoprecipitate contain some factor VIII so could be used if there is a delay in obtaining factor VIII concentrate.

I would aim to use other blood products as indicated by the severity of haemorrhage, in accordance with local and national transfusion guidelines, aiming to give FFP and platelets early in order to reduce potential coagulopathy. I would send intraoperative

samples for full blood count, coagulation studies and fibrinogen, and if blood loss continues I would request fresh frozen plasma and platelets. If the fibrinogen was low I would correct it using cryoprecipitate.

In the case of inadequate haemostasis postoperatively I would be careful to avoid dilutional anaemia, as this is known to impair coagulation and some centres advocate the maintenance of a haemoglobin level of 10 grams per decilitre.

If thromboelastometry were available, I would use it to guide my blood product administration.

Do you know of any drugs that may be useful in the management of this patient?

The recent POISE-3 trial found that tranexamic acid reduced major bleeding significantly (up to 25%) and was safe with a low likelihood of thrombotic events. If not already given I would give a 1g loading dose – if already given I would consider continuing with 1g over 8 hours.

Drugs that may be useful include:

- Desmopressin can be useful in patients with haemophilia. It works by stimulation of V1a receptors, which both improves platelet function and induces endogenous von Willebrand factor and factor VIII production.
- Recombinant factor VIIa can be used in massive haemorrhage to regenerate the thrombin burst.

I would also avoid non-steroidal anti-inflammatory drugs due to their anti-platelet effect.

It would be important to avoid the intramuscular route of administration for any drugs, to avoid intramuscular haematomas.

How else may you limit the need for excessive transfusion in this patient?

Consider pre-op, intra-op and post-op measures.

Preoperatively:

- Avoid excessive use of blood for volume replacement as a young and otherwise fit man is likely to be able to tolerate considerable haemodilution. As far as possible I'd try to reserve transfusion until the surgeon has achieved haemostasis.
- Attempt to correct coagulation abnormalities as much as possible before theatre to reduce bleeding, consider replacement of electrolytes (particularly calcium), correction of acidosis and avoidance of hypothermia.
- Prepare a cell-saver or equivalent autologous transfusion device for use.

At induction and intraoperatively:

- Take special care with intubation so as not to traumatise the airway and cause further bleeding
- Consider the use of a topical vasoconstrictor to reduce nasal bleeding if a nasogastric tube was necessary
- Perform central line insertion under direct ultrasound guidance, avoiding the subclavian route
- Maintain a normal temperature
- Consider intra-operative cell salvage.

Postoperatively:

- Continue care in a high dependency environment with continuous monitoring and regular blood sampling
- Promptly address any abnormalities in coagulation and liaise regularly with haematologists
- Consider accepting a haemoglobin of greater than or equal to 70g/L in accordance with AoA transfusion guidelines.

What is your definition of massive transfusion?

There are many definitions so pick one you know and stick with it.

Massive transfusion is when the equivalent of one blood volume is transfused within 24 hours.

Other possibilities include:

- 50% total blood volume lost in 3 hours
- Bleeding in excess of 150 ml/minute
- In children – transfusion of over 40 ml/kg of red cells.

What are the complications of massive transfusion?

The complications of massive transfusion include those common to any blood transfusion so it is worthwhile learning a list!

Complications of massive transfusion:

- Errors in blood administration – human error, patient identification problems, inadequate cross-checking procedure
- Incompatibility of blood – such as ABO, Rh or other antigens in un-cross-matched blood or patients who have received multiple previous transfusions
- Acute haemolytic reactions
- Anaphylaxis
- Delayed reactions
- fluid overload
- Transmission of infective agents – bacterial, viral or prions
- Citrate related hypocalcaemia
- Hyperkalaemia
- Acidosis
- Dilutional coagulopathy
- Impaired oxygen delivery
- Transfusion-related acute lung injury – recipient neutrophil activation in alveolar membranes
- Disseminated intravascular coagulation
- Hypothermia.

Can you tell me more about thromboelastometry?

Thromboelastometry is becoming increasingly popular and you should be able to describe the technique and give a basic interpretation of the results. To demonstrate a good

A NORMAL THROMBOELASTOGRAPH

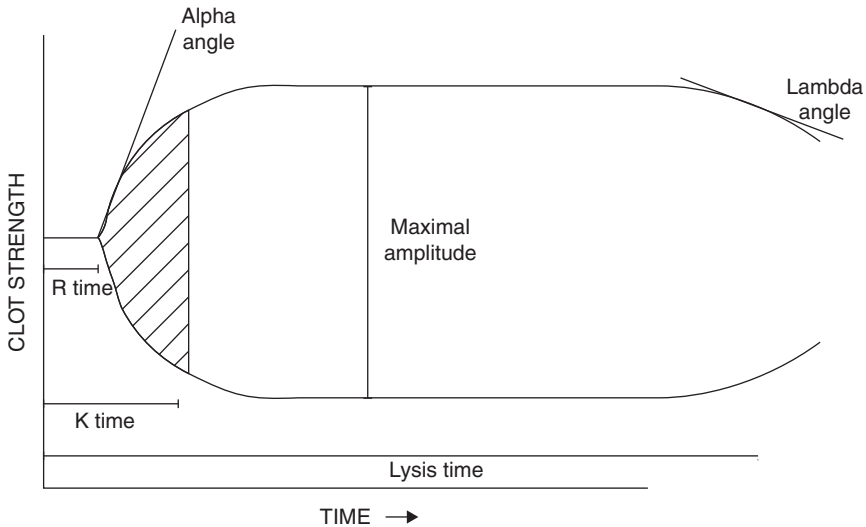


Figure 1.5.3 A normal thromboelastograph.

understanding and help explain your answer it would help to draw a normal trace (Figure 1.5.3, Table 1.5.3).

Thromboelastometry is a bedside test used to assess a patient's coagulation. It involves a pin being rotated in whole blood to analyze the process of coagulation. Clot forms on the pin and a graph is created that shows the change in torque over time. It looks both at the time taken for the thrombus to form, the tensile strength and the elastic properties of the clot. Hospitals may have different machines (i.e. TEG and ROTEM) but both work on similar underlying principals and allow point-of-care testing in a close to real-time fashion.

The measurements and their significance are as follows:

- Time taken to start the clot formation
- Rate of formation
- Maximum strength of the clot
- Time taken for fibrinolysis to start (and its rate and extent).

They can all be used to assess requirement of clotting factors, platelet or fibrinogen replacement.

Can you tell me about another important inherited disorder of coagulation?

There are a number you could mention, but by far the most common is Von Willebrand disease.

Von Willebrand disease is a relatively common disorder of coagulation that is due to either insufficient or ineffective Von Willebrand factor. Von Willebrand factor is a glycoprotein which binds to other proteins in the coagulation cascade (factor VIII).

Table 1.5.3 Thromboelastography measurements and interpretation

Measurement	Explanation	Interpretation
Clotting time or R time	Time from start to curve reaching 1 mm wide Coagulation factor activation	Prolonged in factor deficiencies, with anticoagulants and thrombocytopenia
Clot formation time or K time	Time for the graph to widen from 1mm to 20 mm Coagulation factor amplification	Prolonged in fibrinogen and platelet deficiencies
Maximal clot firmness or maximal amplitude (MA)	Curve width at its widest point	Reduced in fibrinogen and platelet deficiencies
Alpha angle	Line from midline to 1mm point tangential to the curve – indicates speed of clot formation Coagulation factor amplification	Abnormal in clotting factor deficiencies, platelet dysfunction, thrombocytopenia and low fibrinogen
Lysis time	Measured by decrease from maximal amplitude – a decrease in >15% indicates fibrinolysis Lysis is measured at 30 minutes and 60 minutes after MA is recorded	Reduced in poor platelet function and fibrinolysis
Lambda angle	Angle showing lysis rate	

Those with type 1 disorder have only around 75% of normal von Willebrand factor levels; type 2 patients are divided into 3 subtypes and all have qualitative defects in the factor, but may have normal levels; type 3 patients have an autosomal recessive absence of the factor and therefore very severe disease.

Patients are at risk of bleeding from mucosal surfaces which is different from haemophilia in which bleeding is usually into joints or the peritoneal cavity.

If this patient had Von Willebrand disease instead, how might your management have differed?

Preoperatively I would have attempted to ascertain which type of Von Willebrand disease the patient had, and whether he had recent VWF levels checked. Again, I would be in close liaison with a haematologist and seek their advice about timing of administration of drugs and blood products. If they were in agreement I would give 0.3 micrograms per kilogram of DDAVP (desmopressin – which promotes the release of VWF) as soon as possible, as this can help restore clotting function within an hour and has an action lasting about 6 hours.

A normal bleeding time and APTT do not necessarily mean normal VWF levels. If DDAVP is used the coagulation should be checked after an hour but it may be of little help in some subtypes of the disease.

Intraoperatively, I would use either one unit per 5 kilograms of cryoprecipitate, or twenty millilitres per kilograms of fresh frozen plasma, as both of these measures have been shown to raise factor levels by 15% or more.

I would not use factor VIII concentrate, as this contains no von Willebrand factor. Postoperatively DDAVP and blood products should be used to maintain von Willebrand factor levels until the risk of haemorrhage has resolved.

Can you give any examples of disease states that result in bleeding tendencies?

Classify your answer, for example mention diseases causing impairment in coagulation factors, platelets and vascular endothelium.

Disorders that result in clotting factor deficiencies include:

- Advanced liver disease
- Disseminated intravascular coagulation.

Those resulting in platelet dysfunction or reduced numbers of platelets include:

- Disseminated intravascular coagulation
- Haematological malignancy
- Autoimmune platelet diseases
- Splenomegaly
- Uraemia
- Liver disease.

Also there are diseases involving the vascular endothelium, for example:

- Amyloidosis
- Sepsis.

Can you clarify what, in terms of a coagulation disorder, would prevent you from performing regional anaesthesia?

Mention any guidelines you might know about such as the ASRA guidelines for patients receiving regional anaesthesia.

I would avoid regional anaesthesia in the following cases:

- Abnormal APTT, INR or bleeding time, unless the benefits of using it clearly outweighed the risk
- Platelet levels of $80 \times 10^9/L$ or below
- On antiplatelet drugs, or who had stopped taking them less than a week previously, though non-steroidal anti-inflammatory agents are generally considered safe
- Known coagulation disorder, whether or not their laboratory results were normal, as it is possible to have a normal coagulation screen but abnormal coagulation
- Patient received a prophylactic dose of low molecular weight heparin within the last 12 hours
- Patient received a treatment dose of low molecular dose heparin within the last 24 hours.

Following regional anaesthesia, the first postoperative dose of low molecular weight heparin should not be given until 4 hours after the block. Any catheters used should not

be removed until 12 hours after the dose and any subsequent dose should not be given until 2 hours after catheter removal.

Oral anticoagulants, mainly warfarin in this country, should not be given perioperatively where regional anaesthesia is used. Anyone on warfarin preoperatively should stop it long enough in advance for the INR to normalise. It is important to remember that INR mainly assesses factor VII activity and not factors II and X so that a normal INR does not necessarily mean normal coagulation.

Are there any patients in whom you would be particularly concerned about perioperative venous thrombosis?

Categorise into patients with specific risk factors, and patients with specific thrombophilias.

All patients admitted to hospital must undergo a venous thromboembolism risk assessment and this should be documented.

Those at risk include: any patient with:

1. A previous history of thrombosis whether or not they had a proven thrombophilia
2. Diabetes
3. Obesity
4. Malignancy
5. Hyperviscosity syndromes.

Particular thrombophilias that raise alarm because of high thrombotic risk would include:

1. Activated protein C resistance (for example factor V Leiden deficiency)
2. Protein C deficiency
3. Protein S deficiency
4. Antithrombin III deficiency
5. Antiphospholipid syndrome.

I would consult a haematologist about the management of these patient groups.

Those with a high risk of thrombosis should be managed with measures to reduce venous stasis, such as graduated or pneumatic compression stockings, leg elevation, early mobility and leg exercises. Also, intravascular coagulation can be prevented by using drugs such as unfractionated or low molecular weight heparins, or antiplatelet agents.

Further Reading

Blood Transfusion and the Anaesthetist - Red Cell Transfusion 2. *Association of Anaesthetists*. 2008.

Devereaux P, Marcucci M, Painter T. Tranexamic acid in patients undergoing noncardiac surgery *New England Journal of Medicine*. 2022; 386: 1986–1997.

Hebert PC, Wells G, Blajchman MA et al. Canadian Critical Care Trials Group. A multicenter, randomized, controlled

clinical trial of transfusion requirements in critical care. *New England Journal of Medicine*. 1999; 340: 409–417.

Hill SR, Carless PA, Henry DA et al. Transfusion thresholds and other strategies for guiding allogeneic red blood cell transfusion. *Cochrane Database of Systematic Reviews* 2000.

Horlocker T et al. Regional Anesthesia in the Patient Receiving Antithrombotic or Thrombolytic Therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition)

Regional Anesthesia and Pain Medicine:
January/February 2010; 35 (1): 64–101

Shah U, Narayanan M, Smith J. Anaesthetic considerations in patients with inherited disorders of coagulation. *BJA Education*. 2014; available from <https://academic.oup.com/bjaed/article/15/1/26/257411>

www.transfusionguidelines.org.uk/docs/pdfs/htm_edition-4_all-pages.pdf

Stainsby D, MacLennan S, Thomas D, Isaac J, Hamilton PJ. Guidelines on the management of massive blood loss. *British Journal of Haematology*. 2006; 135: 634–641.

1.5.4 Anticoagulant, Antiplatelet and Anti-fibrinolytic Agents – Benjamin Hofland-Ward, Alexandra K Freeman and Susanna T Walker

Can you classify anticoagulants?

Remember, as with everything, you should classify the drugs if possible as you will be less likely to forget any.

Anticoagulant drugs can be classified into those that inhibit platelet function, those that affect the clotting cascade, and those that lead to the breakdown of a clot once it has formed.

Let's start by talking about antiplatelet agents. Can you tell me about all the drugs you know of that inhibit platelet function?

There are several drugs that affect platelet function, all of which work in slightly different ways. It is helpful to have an understanding of platelet function to understand how these drugs work.

Platelets are activated when they are exposed to the subendothelial matrix of a damaged vessel wall. Activation leads to degranulation of the platelets and then a process of aggregation where platelets stick to each other, and clot formation follows. Von Willebrand factor and factor VIII are essential for the initial phase of platelets sticking to the vessel wall. The degranulation process leads to release of thromboxane A₂, which increases platelet aggregation and also causes localised vasoconstriction of the vessel. Distal to the clot, vessel walls release prostacyclin, which has the opposite effect to thromboxane A₂, preventing platelet aggregation and leading to vasodilatation. This aims to localise and minimise the clotting process.

Thrombin, produced as part of the clotting cascade, increases the activity of platelets by increasing the production of glycoprotein IIb/IIIa receptors. These receptors are required for binding of fibrinogen to platelets, leading to cross-linking of platelets, and strengthening the clot formed.

Adenosine diphosphate (ADP) is also released from the platelets in the degranulation process. This has a positive feedback effect on the platelets by binding to an ADP receptor on the platelet surface. This increases platelet activity by enabling the glycoprotein IIb/IIIa receptor to transform into its active form and therefore facilitate binding of fibrinogen to the platelets (Figure 1.5.4.1).

Drugs that affect platelet function include:

1. **Non-steroidal anti-inflammatory drugs (NSAIDs)** e.g., aspirin. These work by inhibiting cyclo-oxygenase enzymes, therefore reducing production of thromboxane

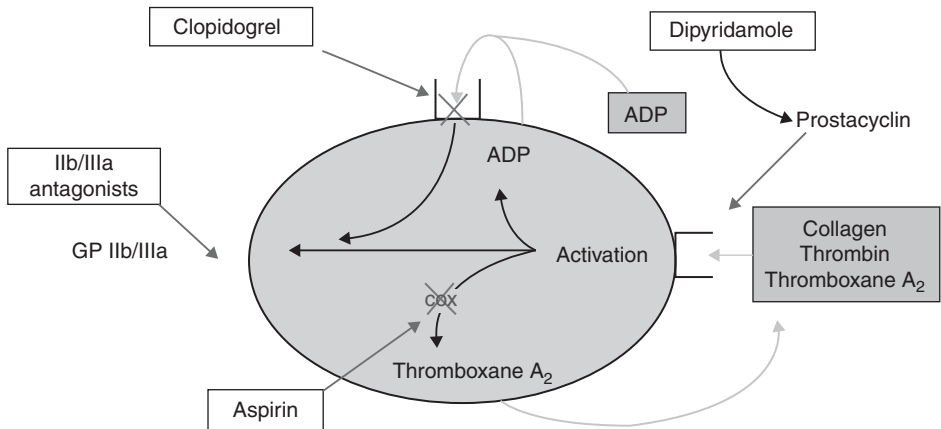


Figure 1.5.4.1 Sites of action of antiplatelet agents.

A₂ from arachidonic acid, thus inhibiting platelet aggregation and preventing localised vasoconstriction of the vessel.

2. **Clopidogrel.** This works by irreversibly blocking the ADP receptor and therefore indirectly reduces platelet activity and inhibits activation of the glycoprotein IIb/IIIa receptor.
3. **Dipyridamole.** This inhibits platelet phosphodiesterase, which normally breaks down cyclic AMP within the platelets. High levels of cyclic AMP prevent activation of platelets, thereby reducing platelet adhesion to vessel walls. It also increases the effects of prostacyclin, thus further preventing platelet aggregation, and leading to vasodilatation.
4. **Abciximab.** This is a synthetic antibody, which binds strongly to the glycoprotein IIb/IIIa receptor, therefore preventing binding by fibrinogen.
5. **Prostacyclin.** As previously mentioned, this is produced endovascularly leading to localised vasodilatation and inhibition of platelet aggregation. It can be given as a continuous infusion to anticoagulate a haemofiltration circuit in a patient unable to receive heparin. It may also be used for its vasodilating properties, such as in patients with Raynaud's disease.
6. **Dextrans.** These are large chain carbohydrate molecules, which may be given for fluid resuscitation. They have an inhibitory effect on von Willebrand factor and therefore have an anticoagulatory effect.

Can you describe, with the help of a diagram if you wish, exactly how anti-inflammatory drugs work to inhibit platelet function?

It's always worth drawing a quick diagram if you can, but make sure you make it a big, clear diagram (Figure 1.5.4.2).

Membrane phospholipids are broken down to arachidonic acid by phospholipase A. This is then converted to prostaglandin H₂ by cyclo-oxygenase enzyme, and then converted on by a variety of enzymes to thromboxane A₂ in platelets, prostacyclin (PGI₂) in the vascular endothelium and various prostaglandins (PGE₂, PGF_{2α}, PGD₂) at other

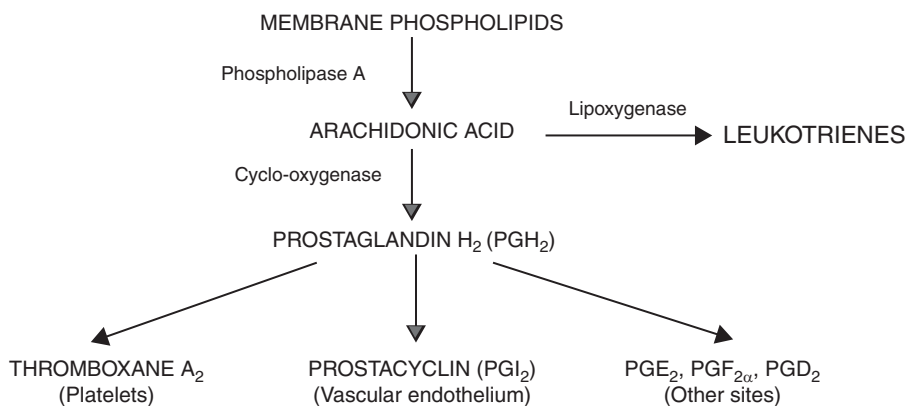


Figure 1.5.4.2 Synthesis of prostaglandins.

sites. There is an alternative pathway enabling arachidonic acid to be converted to leukotrienes by lipoxigenase enzyme, which is not affected by NSAIDs. It is thought that a relative excess of leukotrienes due to an inhibition of cyclo-oxygenase may be the cause for the wheeze precipitated by NSAIDs.

What is the difference between aspirin and other NSAIDs with regards to duration of action?

Aspirin leads to an irreversible inhibition of the cyclo-oxygenase enzyme, whilst other NSAIDs lead to reversible inhibition. This therefore means that the effect of aspirin lasts for the lifetime of the platelet, whilst the other NSAIDs only have an effect whilst the drug is in the system in its active form. When given in a low dose, aspirin specifically has an effect on platelet cyclo-oxygenases but not on vessel wall cyclo-oxygenase, therefore having an anticoagulant effect on platelets, whilst retaining the production of vessel wall prostacyclin.

Which groups of patients are routinely started on aspirin and clopidogrel or other antiplatelet agents?

Aspirin and clopidogrel are most commonly used for their anti-platelet effect in patients who are at risk of myocardial infarction or cerebrovascular accident. Both these drugs are routinely started in patients who have had percutaneous coronary intervention with insertion of a coronary stent.

There are two types of stent that are commonly used – drug-eluting stents and bare-metal stents. Both types lead, in the initial stage, to bare metal being exposed in the coronary vessel, which is highly thrombogenic and prone to in-stent thrombosis. Prevention of this is therefore extremely important. Drug-eluting stents have the effect of reducing intimal proliferation, and are therefore used to reduce the rates of in-stent re-stenosis. However, the drug that is released has the effect of prolonging the time that bare metal is exposed, therefore increasing the risk of in-stent thrombosis for a longer period. It has been suggested that dual anti-platelet therapy should be continued for at least a year in patients with drug-eluting stents. A study has shown that premature

cessation of anti-platelet therapy is the strongest predictor of subsequent stent thrombosis. The overall recommendations are that if non-cardiac surgery is known to be necessary within a year of stent insertion then a bare-metal stent should be used in preference to a drug-eluting stent. In a patient who has recently had a stent inserted, elective non-cardiac surgery should be avoided if possible until clopidogrel can be safely stopped. In situations where surgery cannot be delayed careful planning and communication with the cardiology team is required.

Can you now tell me about the drugs that exert an effect on the clotting cascade that you mentioned in your initial classification?

Once again, it is easier to understand how these drugs work by having an understanding of the clotting cascade (Figure 1.5.4.3). Try and describe the clotting cascade and then relate the drugs to this when discussing how they work.

The classical description of the clotting cascade consists of a series of clotting factors that are present within the plasma in an inactive state. Activation of one leads to activation of the next in a cascade fashion. Various co-factors are required at certain stages of the pathway. The purpose of the cascade is to produce thrombin, which converts fibrinogen into fibrin, leading to clot formation. The clotting cascade is classically quoted as having two limbs – the intrinsic pathway and the extrinsic pathway. These then converge to form the final common pathway. The intrinsic pathway consists of factors XII, XI and IX, and is activated by blood coming into contact with subendothelial connective tissues. The extrinsic pathway consists of factors III and VII and is activated by tissue damage much more rapidly than the intrinsic pathway. Its purpose is to help promote activation of the intrinsic pathway.

The final common pathway starts by activation of factor X, which occurs as the end result of both pathways. Activated factor X converts pro-thrombin to thrombin, which in turn converts fibrinogen to fibrin, as already mentioned.

Drugs that affect the clotting cascade can be classified into:

1. Vitamin K antagonists, which would include warfarin and phenindione
2. Factor Xa inhibitors, which can be subdivided into:
 - 2.1. Unfractionated heparin
 - 2.2. Low molecular weight heparins, such as enoxaparin
 - 2.3. Oligosaccharides, such as fondaparinux
 - 2.4. Heparinoids, such as danaparoid sodium
 - 2.5. Direct oral factor Xa inhibitors (DOACs), such as apixaban and rivaroxaban
3. Direct thrombin inhibitors, which can be subdivided into:
 - 3.1. Bivalent compounds, such as lepirudin
 - 3.2. Univalent compounds, such as argatroban.

Tell me how warfarin works.

Warfarin and phenindione are both coumarin derivatives. Coumarin is a naturally occurring compound found in some plants that is toxic and was noted to have anticoagulant properties.

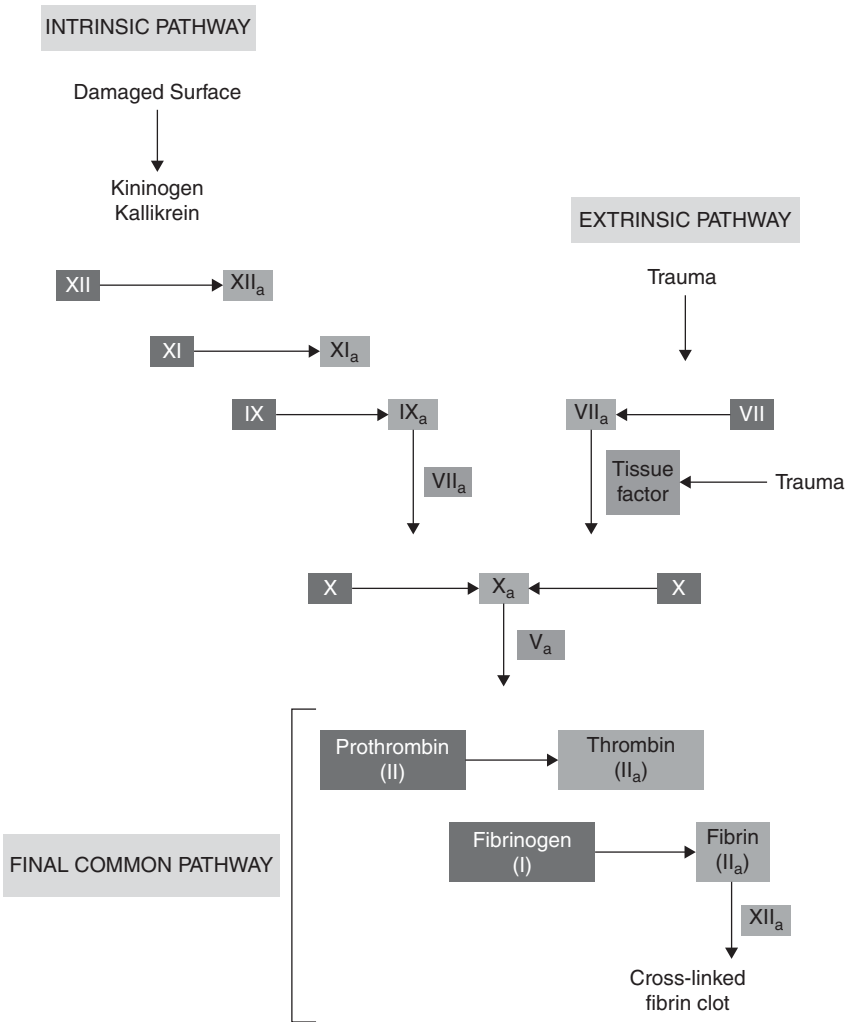


Figure 1.5.4.3 The classical clotting cascade.
Adapted from Anaesthesia UK

Warfarin works by inhibiting the reduction of vitamin K. Vitamin K is required in its active, reduced form as a co-factor in the γ -carboxylation of glutamic acid residues in the production of factors II, VII, IX and X in the liver. Inhibition of vitamin K production therefore leads to inhibition of clotting factor production. As this process requires inhibition of the synthesis of new clotting factors, warfarin may take up to 72 hours to have a full effect on the clotting cascade.

The effects of warfarin need to be carefully monitored on a regular basis by checking a patient's prothrombin time, or INR (international normalised ratio).

It is important to note that, since warfarin is metabolised by the liver, factors leading to liver enzyme induction or inhibition will have an effect on the levels of warfarin. For example, alcohol, and some antibiotics such as erythromycin lead to an inhibition of

metabolism and therefore an increase in warfarin levels. Conversely, drugs such as barbiturates and carbamazepine induce liver enzymes and therefore reduce the levels of warfarin.

How does heparin work? What is the difference between unfractionated heparin and low molecular weight heparin?

Heparin is a naturally occurring sulphated glycosaminoglycan, which has been found in various sizes within mast cells and liver cells. Heparin has its effect by increasing the activity of anti-thrombin III (a naturally occurring inhibitor of the clotting cascade) by up to 1,000 times. When anti-thrombin III binds to thrombin, an inactive complex forms. Antithrombin III also inactivates other factors including II, IX, X, XI and XII. Unfractionated heparin inhibits factor Xa at low concentrations, but will also inhibit these other factors as blood concentrations rise. It is usually administered as a continuous infusion (although occasionally as subcutaneous injections) to achieve therapeutic blood levels, and its effect wears off within about 4 hours of stopping the infusion. Monitoring of the activated partial thromboplastin time (APTT) is required to adjust the infusion rate. This is a measure of the intrinsic clotting cascade.

Low molecular weight heparins, such as enoxaparin and tinzaparin, are the smaller weight fractions (ranging from 2000 to 8000 daltons) of the heparin. They are more effective at inhibiting factor Xa than unfractionated heparin, and have minimal effect on other factors. For this reason, their effectiveness cannot be measured using the APTT. Levels of factor Xa can be measured, although this is not routinely done. They have the benefit over unfractionated heparin of having a longer half-life, and so can be given as a once daily injection.

What can you tell me about heparin-induced thrombocytopenia?

If you're asked about this you're doing well!

There are two causes of thrombocytopenia related to the use of heparin. These can be divided into type I and type II.

Type I is a non-immune mediated thrombocytopenia, which is reasonably common following the use of heparin. It does not usually cause any problems, and the platelet count usually recovers quickly even when the heparin isn't stopped.

Type II, however, is an immune-mediated process with much more serious consequences. It usually occurs after exposure to unfractionated heparin, although it may also be associated with use of low molecular weight heparin. It occurs when heparin complexes with platelet factor 4, forming antigenic material to which antibodies form. Binding of the antibodies leads to platelet activation, generation of thrombus, and a resultant thrombocytopenia. This therefore causes a pro-thrombotic state, and so these patients are at risk of severe thrombotic events such as stroke, PE, MI and limb ischaemia. The platelet count rarely drops very low, and therefore patients are less at risk of bleeding. This condition may occur 5–15 days after heparin has been given, even if it is no longer being administered. It is important to consider the condition in patients with an unexplained thrombocytopenia, in patients with thrombosis associated with thrombocytopenia, and in those with necrotic lesions at the site of previous heparin injections.

If a patient is suspected of having heparin-induced thrombocytopenia (HIT), a 'HIT Screen' can be sent to look for immune complexes. The result from this can take some

days, so in the meantime the patient should have any heparin or low molecular weight heparin stopped. An alternative anticoagulant should be started, such as a direct thrombin inhibitor (lepirudin or argatroban), or an alternative factor Xa inhibitor (danaparoid sodium or fondaparinux). Once the platelet count has recovered to acceptable levels, warfarin therapy can be considered for long-term anticoagulation if required.

What is danaparoid sodium?

Danaparoid sodium is a low molecular weight heparinoid and is effective first line treatment for heparin-induced thrombocytopenia.

Unlike low molecular weight heparin (LMWH), danaparoid is very unlikely to react with heparin-associated anti-platelet antibodies, and so does not cause the HIT syndrome itself. Like LMWH, its activity can be monitored by measuring plasma factor Xa levels.

What are DOACs?

Direct oral factor Xa inhibitors (DOACS) such as apixaban and rivaroxaban are newer anticoagulants that work by inhibiting both free and clot bound factor Xa. These drugs have been approved for use in multiple thromboembolic disorders, including reduction of stroke risk in non-valvular atrial fibrillation, thromboprophylaxis following hip or knee replacement surgery, the treatment of deep vein thrombosis or pulmonary embolism, and prevention of recurrent deep vein thrombosis and pulmonary embolism. This avoids the use of injection with LMWH and also the continuous monitoring requirements caused by the narrow therapeutic window of warfarin. Therefore, they are more popular choices for patients, and increasingly commonly prescribed.

Recent advances have also meant that DOAC agents can be rapidly reversed, using a drug called andexanet alfa (Ondexxya). This drug may be used specifically in situations of life-threatening or uncontrolled bleeding. It is a recombinant, modified human factor Xa decoy protein, which binds specifically to apixaban or rivaroxaban. This then restores endogenous factor Xa thereby reversing their anticoagulant effects. Currently recommendations exist for routine use in gastrointestinal bleeding and in research settings only in intracranial haemorrhage.

What recommendations do you know of regarding patients taking anticoagulant medications and performing a central neuraxial blockade?

It is relatively common for patients receiving some form of anticoagulation to require a central neuraxial blockade for surgery. Clearly a patient that is fully anticoagulated with any form of anticoagulant should not receive a neuraxial block, as the risk of epidural haematoma formation would be too great.

For patients receiving warfarin, it is generally accepted that an epidural can be sited if the INR is less than 1.5.

For patients receiving unfractionated heparin as an infusion, at least 4 hours should have elapsed between stopping the infusion and placing an epidural catheter.

For patients receiving low molecular weight heparin, at least 12 hours should have passed since the last dose before inserting, or removing, an epidural catheter. Another

2 hours should pass after inserting or removing an epidural catheter before giving the next dose of low molecular weight heparin.

For surgery where large doses of heparin may be given intraoperatively, such as vascular surgery, some anaesthetists are cautious about siting an epidural catheter. However, large observational studies have shown no increased incidence of epidural haematoma formation.

Finally, many patients come into hospital taking anti-platelet agents or DOACs. It is generally accepted that a patient taking low dose aspirin can have a neuraxial block without the need to stop taking aspirin. However, there is more concern about the use of clopidogrel. Most anaesthetists would agree that ongoing clopidogrel use would contraindicate the placing of an epidural. It should have been stopped for at least 7 days before attempting to site a central neuraxial block to minimise the risk of epidural haematoma. For DOACs, recommendations vary depending on the drug used, and a degree of caution should occur in patients with a reduced creatinine clearance. When low doses of DOACs are used, the last intake should be a minimum of 24 hours for rivaroxaban and edoxaban, 35 hours for apixaban, and 48 hours for dabigatran before neuraxial procedures.

In any patient with a degree of anticoagulation, it is much safer to perform a single shot spinal technique than a technique involving the much larger epidural needle. This should therefore be considered, if appropriate.

Are you aware of any methods available for checking a patient's coagulation status?

There are a multitude of tests that can be performed to assess a patient's coagulation status. These can be divided into lab-based tests, and near-patient tests.

The lab-based tests would include:

1. **INR (international normalised ratio).** As already mentioned, this is a measure of the extrinsic pathway, and is used predominantly as a measure of warfarin activity.
2. **APTT (activated partial thromboplastin time).** This has also been mentioned earlier and is a measure of the intrinsic pathway. It is used as a measure of heparin activity.
3. **Thrombin time.** This is a measure of the final common pathway. It will be affected in patients with abnormal or deficient thrombin.
4. **Platelet count.** Coagulation may be deficient if a patient has a low platelet count. Preoperatively we would generally aim for a platelet count of greater than $100 \times 10^9/L$.
5. **Platelet function assay.** This assesses platelet function. Patients with a normal platelet count can have abnormal clotting if their platelets are not functioning normally. Platelet function may be affected by anti-platelet agents, hereditary diseases, or other factors, such as a very high serum urea.

The near-patient tests would include:

1. **Bleeding time.** This is performed by making a standardised incision and timing how long it takes for clot formation. This is predominantly an assessment of platelet function.
2. **Activated clotting time (ACT).** This is most commonly measured in vascular and cardiac theatres. It is a quick assay of clotting that measures time for clot formation.

A normal result would be 100–150 seconds. Prolonged values may represent abnormal clotting as a result of heparin given intraoperatively, or a coagulopathy.

3. **Thromboelastogram (TEG).** This is another quick test that can be performed in theatre or on the ICU if the correct equipment is available. It gives an indication of time to clot activation which acts as a surrogate measure of clotting factors; strength of clot which acts as a measure of platelet function; and breakdown of clot, which may be more rapid than normal in conditions such as DIC. This can therefore be used to tailor the coagulation factor replacement to the defect detected. There are some newer reagents that are used with the TEG that can enable near-patient assessment of platelet dysfunction as a result of anti-platelet agent use.

Finally, can you tell me about the fibrinolytic agents?

The fibrinolytic agents are plasminogen activators. During the process of clot breakdown, inactive plasminogen has to be converted to the active plasmin, which can then break fibrin down to fibrin degradation products, thus dissolving the clot. Fibrinolytic agents initially form a fibrino–plasminogen activator complex. This then goes on to convert further plasminogen molecules to active plasmin. There are many examples of fibrinolytics including streptokinase and alteplase. They are generally given to dissolve clot in a life-threatening situation such as a myocardial infarct or pulmonary embolus. They carry with them the risk of serious bleeding, and so there are situations where the risk of administration outweighs any potential benefit of treatment.

You review a patient in the preoperative assessment clinic who is awaiting an elective hemicolectomy for diverticulitis. They are taking rivaroxaban for chronic atrial fibrillation. What are your considerations regarding their anticoagulation and how will you manage these?

For any patient requiring surgery who is taking anticoagulant agents, the risk of bleeding must be balanced against the risk of thrombosis. For this reason, I would consider each case individually, in collaboration with the surgeon and haematologist.

Risk of bleeding would depend on the procedure, anticoagulant agents and any additional patient risk factors. Patients awaiting elective surgery will require their rivaroxaban to be paused to allow sufficient time for the anticoagulant effects to be minimised. An interval of 24 hours in normal renal function and 48–72 hours in impaired renal function is usually acceptable. These time intervals would also allow the performance of neuraxial or regional anaesthetic procedures.

Risk of thrombosis would depend primarily on the indication for anticoagulation. Patients at high risk may warrant bridging therapy with unfractionated or low molecular weight heparin. Risk of thromboembolism in AF can be calculated using the CHA₂DS₂ VASc score, with a score of 7–9 denoting high risk.

With regard to this patient, assuming they had normal renal function and the surgery was deemed to have a low bleeding risk, the rivaroxaban should be paused for 24 hours preoperatively. I would, therefore, advise the patient to omit their rivaroxaban on the day prior to, and the day of, the procedure. No bridging therapy would be required.

Postoperatively, the patient should be prescribed prophylactic low molecular weight heparin until the surgical team are happy for full anticoagulation to be recommenced.

How would you manage this patient if they presented for emergency surgery?

As in the elective setting, I would balance the risk of bleeding with the risk of thrombosis, in conjunction with the surgeon and haematologist.

I would ascertain the timing of the last dose of the rivaroxaban and ensure the patient did not receive any further anticoagulant agents. Ideally, the surgery would be delayed until 24 hours after the last dose of rivaroxaban. If this was not possible, I would administer tranexamic acid 1g IV prior to skin incision. I would avoid any regional or neuraxial procedure, as well as any drugs that could affect haemostasis (e.g. NSAIDs and colloids).

If the patient was at significant bleeding risk, I would liaise with the haematologist regarding use of prothrombin complex concentrate at a dose of 25–50 units/kg.

You may be aware of specific reversal agents for the direct oral anticoagulants (e.g. andexanet for rivaroxaban and apixaban). At the time of writing, the recommendation for the use of these agents in the perioperative setting has been withdrawn by the British Society of Haematology due to insufficient evidence to support their use.

How would your management differ if this patient was taking warfarin rather than rivaroxaban?

Warfarin has a longer half life (approximately 36 hours) than the direct oral anticoagulant agents. It also has the benefit of therapeutic monitoring, in the form of INR.

In the case of elective surgery, I would advise the patient to stop their warfarin 5 days prior to their procedure and check their INR on admission to ensure resolution of anticoagulant activity; an INR of <1.4 is usually acceptable.

In the case of emergency surgery, I would stop any anticoagulant agents immediately and check the patient's INR both via an urgent laboratory sample and, if available, a point-of-care test. Assuming the patient's INR was within the therapeutic range, I would administer 5 mg of IV vitamin K. If the surgery had a high risk of bleeding or if immediate reversal was required, I could administer prothrombin complex concentrate at a dose of 25–50 units/kg.

With either reversal strategy I would recheck the INR at the start of surgery and appropriate time intervals during the procedure and postoperative period. I would liaise with the surgeon regarding postoperative thromboprophylaxis until full anticoagulation could be resumed.

Further Reading

Howard-Alpe GM, de Bono J, Hudsmith L, et al. Coronary artery stents and non-cardiac surgery. *British Journal of Anaesthesia*. 2007; 98(5): 560–574.

Keeling D, Campbell TR, Watson H. Association of Anaesthetists of Great

Britain and Ireland, Obstetric Anaesthetists' Association and Regional Anaesthesia UK. Regional anaesthesia and patients with abnormalities of coagulation. *Anaesthesia*. 2013; 68: 966–972.

Peri-operative management of anti-coagulant and anti-platelet therapy. *British Journal of Haematology*. 2016; 175: 602–613.

1.5.5 Thromboembolism – Steffan Morgan and Ami Jones

An orthopaedic senior house officer asks you what factors increase the risk of a patient suffering from a venous thromboembolism as he is unsure whether or not he should prescribe prophylaxis for one of his patients.

Classify into patient factors and surgical factors – there is NICE guidance on this subject, of which you should be aware.

Patient factors:

- Active cancer or treatment
- Age > 60
- Dehydration
- Known thrombophilias
- Obesity (BMI <30 kg/m²)
- Significant medical comorbidities
 - e.g. heart/respiratory/metabolic or endocrine disease
- History or family history of VTE
- Use of hormone replacement therapy
- Use of oestrogen containing contraception
- Varicose veins with phlebitis
- Pregnancy or < 6 weeks postpartum.

Surgical factors:

- Significantly reduced mobility for > 3 days
- Hip or knee replacement
- Hip fracture
- Anaesthetic and surgical time > 90 minutes
- Lower limb surgery with anaesthetic and surgical time > 60 minutes
- Inflammatory intra-abdominal condition
- Critical care admission.

This should be balanced against the risk factors for bleeding which can also be categorised into patient and surgical.

Patient factors:

- Active bleeding
- Acquired bleeding disorders e.g. acute liver failure
- Use of anticoagulants
- Acute stroke
- Thrombocytopenia (platelet count < 75 × 10⁹/L)
- Uncontrolled systolic hypertension (>230/120 mmHg)
- Inherited bleeding disorders (e.g. haemophilia).

Admission related:

- Neurosurgery/spinal surgery/ophthalmic surgery
- Surgery with high bleeding risk

- Lumbar puncture/epidural/spinal anaesthesia within the next 12 hours
- Lumbar puncture/epidural/spinal anaesthesia within the previous 4 hours.

What can be done to reduce a patient's risk of suffering from a venous thromboembolism?

You might want to divide your answer into physical and mechanical methods or pre-, intra- and post-op.

Preoperatively all patients should be risk-stratified for VTE on admission or in the outpatient setting this should involve an MDT approach.

Options for mechanical prophylaxis include:

- Anti-embolic stockings
- Intermittent pneumatic compression
- Foot impulse devices.

Options for pharmacological prophylaxis include:

- Unfractionated heparin
- Low-molecular weight heparin
- Warfarin
- Direct factor Xa inhibitors
- Aspirin
- Danaparoid
- Fondaparinux
- Lepirudin
- Dextrans.

Intraoperatively it is important to keep the patient well hydrated and pay careful attention to positioning. Whilst on the operating table intermittent pneumatic compression devices can be applied to the calves for the duration of the operation.

Regional anaesthetic techniques have been shown to reduce the risk of VTE.

Postoperatively keeping the patient well hydrated and encouraging early mobility reduces risk of VTE.

How would you classify the risk of venous thromboembolism in a 58-year-old obese woman, with a history of breast cancer 5 years previously who is undergoing total knee replacement?

This patient has risk factors given that she is obese with a history of malignancy and is undergoing lower limb arthroplasty. As she is undergoing knee arthroplasty, tourniquet-induced venous stagnation may also be an issue.

What prophylaxis might you provide her with?

She should receive both mechanical prophylaxis with compression stockings and receive LMWH at a time suitable to allow for safe regional anaesthesia.

What prophylaxis might you prescribe for a 23-year-old woman who is fit and well, of a normal BMI, undergoing an elective excision of an ovarian cyst?

The patient is low risk and is undergoing a low-risk procedure, therefore good hydration, early mobilisation and thrombo-embolic deterrent (TED) stockings would be appropriate measures.

You are asked to assess a 35-year-old man in A&E who had a DVT confirmed 2 days ago. He is complaining of chest pain and shortness of breath and has oxygen saturations of 92% on room air. The emergency medicine registrar is concerned that he is suffering from a pulmonary embolism (PE).

What clinical features may support this diagnosis?

Clinical symptoms of pulmonary embolism include:

- SOB
- Pleuritic chest pain
- Apprehension
- Cough
- Haemoptysis
- Leg pain/diagnosed DVT
- Collapse
- Cardiovascular collapse.

Clinical signs include:

- Pale mottled skin
- Tachypnoea
- Tachycardia
- Signs of DVT
- Altered consciousness
- Raised JVP
- Parasternal heave
- Loud P2
- Central cyanosis.

Baseline blood tests have been sent to the laboratory, what results would support a diagnosis of PE?

- Raised white cell count
- Positive D-dimer has good sensitivity but poor specificity
- Raised troponin
- Raised liver transaminases
- Arterial blood gases demonstrating a reduced PaO₂.

What might his ECG and chest X-ray show?

Please note that the ECG and chest X-ray can often be normal.

ECG changes:

- Sinus tachycardia
- Right axis deviation
- Right bundle branch block
- Atrial fibrillation
- T-wave inversion anteriorly ST abnormalities
- Right ventricular strain pattern of S1 Q3 T3.

Chest X-ray changes:

- Mostly done to exclude other pathology
- Hilar dilatation
- Wedge density (pulmonary infarct)
- Pulmonary oligoemia if the PE is massive (watermarks sign).

What tests could be arranged to confirm the diagnosis?

There are a number of tests that aid diagnosis of PE:

- Computerised tomographic pulmonary angiography (CTPA) is the most accessible and effective form of imaging.
- Invasive pulmonary angiography is rarely performed and is usually reserved for patients in whom CTPA is inconclusive.
- Ventilation–perfusion scans are another form of diagnostic imaging which can be employed with reasonable accuracy, but this tends to be used in patients in whom a CT scan is not ideal. Although CTPA is safe in pregnant women concerns have arisen regarding exposure of breast tissue to ionising radiation.
- Echocardiography can also be used to rapidly evaluate a patient who is shocked to evaluate the likelihood of PE.
- MR angiography avoids ionising radiation although technically difficult and insufficient sensitivity.
- Ultrasound of leg veins in patients with symptoms of leg DVT has good sensitivity and specificity if radiation may need to be avoided. Anticoagulation can be commenced with a positive finding.

What might an echo show in a patient with a massive PE?

An echo classically shows:

- Dilated hypokinetic right ventricle
- Dilated pulmonary artery
- Paradoxical septal shift or so called kissing ventricles
- Tricuspid regurgitation
- Right-sided pressure–volume overload
- Occasionally proximal thrombus visible in the main pulmonary artery.

What would be appropriate treatment for this patient?

Management of pulmonary embolism should be based on the severity. This can be divided into massive, sub-massive and non-massive. Massive PE with a

haemodynamically unstable patient should be managed with thrombolysis or embolectomy. Sub-massive PE with a haemodynamically stable patient and evidence of right ventricular dysfunction should strongly consider thrombolysis/embolectomy, but this needs to balance with risk of bleeding. Non-massive PE in which the patient is haemodynamically stable, with normal RV function, should be anticoagulated with low-molecular weight heparin. This can be given prior to diagnostic testing if clinical suspicion is high and in the absence of significant bleeding risk. Patients are often switched to DOACs or warfarin on discharge.

As you are on the phone to your consultant the nursing staff call you over urgently as the patient has become unresponsive and looks deeply cyanosed. Describe your initial actions.

I would assess his airway, breathing and circulation and call for further assistance. I would open his airway and give 100% oxygen via a non-rebreather mask and if he was making no respiratory effort I would commence manual ventilation. If no pulse was palpable I would commence external cardiac compressions as per the current ALS guidelines.

Would his arrest alter how you would treat his PE?

This patient fulfills the criteria of massive PE and requires immediate intravenous thrombolysis with a bolus followed by an infusion of alteplase, providing he has no contraindications to thrombolysis. He will also require endotracheal intubation, ventilation, fluid resuscitation and line insertion. Following thrombolysis the patient should be commenced on unfractionated heparin (where reversal may be required) or low molecular weight heparin.

The patient does well and makes a full recovery. What tests might he need to undergo following his discharge from hospital?

He needs investigation of potential inherited thrombophilic disorders such as factor V Leiden, protein C&S deficiency and anti-thrombin deficiency.

You are asked to review a 24-year-old man in recovery who has just undergone intramedullary nailing of a fractured femur. Nursing staff are concerned because he is desaturating to 88% when he takes his oxygen off and he is quite confused, even though it has been almost an hour since he woke from his general anaesthetic.

Outline your initial assessment of this patient.

Follow a structure for the assessment of any patient in the recovery room.

I would ensure that the patient's airway, breathing and circulatory status were adequate, that he was receiving supplementary oxygen and had IV access. I would then make an assessment of his conscious level using the Glasgow Coma Scale (GCS) and ask to check the patient's blood sugar.

Once I was sure that his vital signs were sufficient I would move on to review the anaesthetic chart making particular note of any pre-morbid conditions or physiological abnormalities that were present preoperatively. I would ascertain what type of

anaesthetic the patient underwent, whether any regional techniques were employed, what drugs and fluids were administered to him and assess how stable the patient had been intraoperatively as well as making a note of any untoward incidents. With the anaesthetic being fully reviewed, possible causes of the patient's clinical condition may become more apparent, for example a high spinal block or incomplete reversal of neuromuscular blockade. As this patient has sustained a long bone fracture, I would examine the admission notes to see if there were any other significant injuries sustained which may be of relevance to his current condition.

The patient has sats of 97% on 8 litres of oxygen, and his airway is patent. His respiratory rate is 30 breaths per minute, his pulse rate 120 beats per minute and his blood pressure 130/80. His GCS is 13 as he is confused and is opening his eyes to voice and moving all four limbs appropriately. He was previously fit and well and broke his femur playing football with no other injuries sustained. He received a general anaesthetic alone along with 2 litres of crystalloid and 15 mg of morphine, paracetamol and a non-steroidal anti-inflammatory drug. The anaesthetic proceeded uneventfully, the procedure taking 70 minutes in total. You note that his saturations were 95% preoperatively on room air.

What is your differential diagnosis?

Be systematic.

The patient is tachycardic, tachypnoeic, has a moderate oxygen requirement and is confused.

He may have been slightly hypoxic prior to surgery. Working through systems, his respiratory compromise could be due to basal atelectasis or retained secretions, pneumonia, fat embolus, aspiration pneumonitis, pulmonary embolus (although this would not be high on my differential diagnosis list as he had only recently been admitted to hospital), drug or transfusion reaction.

His altered conscious level could be due to the effects of general anaesthesia, hypoxia, hypercapnoea, a coexisting undiagnosed head injury, fat embolism, excessive opiates, sepsis, meningitis, an undiagnosed ketoacidosis or a hypoglycaemia.

Other conditions such as stroke, intracerebral haemorrhage or space occupying lesion would be much less likely given his age and premorbid state.

A pre-existing chest infection exacerbated by a general anaesthetic or a fat embolism could account for these symptoms and signs.

What do you think is the likely cause and what other tests or examinations would you want to perform to confirm this?

Given the history of a long bone fracture and subsequent fixation and the combination of respiratory difficulties and neurological impairment, I think that the most likely diagnosis would be fat embolism syndrome.

The triad of pulmonary, neurological and cutaneous signs is a common presentation. Gurd's criteria for diagnosis can also be used with one major and four minor criteria required to satisfy diagnosis (see below for details). I would examine the patient for a petechial rash on the trunk or upper limbs. I would also perform a full respiratory and neurological examination on the patient to rule out other focal abnormalities. I would

take blood for arterial blood gas analysis as well as a full blood count, U&Es, a CRP, liver function tests and a coagulation profile. Anaemia, thrombocytopenia, hypoalbuminaemia, hypocalcaemia and raised serum lipase have all been reported. I would ask the hospital laboratory whether they were able to perform analysis of blood, sputum and urine for fat globules as these are classically said to be present although there is no evidence that they are truly diagnostic. I would also order a chest X-ray and an ECG as fat embolism can result in bilateral hazy consolidation and evidence of right heart strain. I would consider imaging the patient's brain to rule out other causes of acute confusion. Fundoscopy in this patient may show retinal haemorrhages. If neurological signs are present an MRI of the brain may show multiple non-confluent hyperintense lesions scattered throughout grey and white matter. Fat emboli can occasionally be visualised by transoesophageal ultrasound, transcranial doppler and peripheral venous duplex scanning.

Gurd's criteria:

Major criteria

- Axillary or subconjunctival petechia
- Hypoxaemia with bilateral radiological changes
- Cerebral signs unrelated to head injuries.

Minor Criteria

- Tachycardia
- Pyrexia
- Retinal emboli on fundoscopy
- Fat in urine or sputum
- A sudden decrease in haematocrit and platelets
- Increasing ESR.

If all tests point to the diagnosis of fat embolism being correct, how would you manage this patient?

The main approach to managing a patient with fat embolism is supportive and these patients are often managed in a critical care setting. They may require intubation and ventilation and are at risk of developing ARDS. The main aims of neurological treatment are to prevent secondary brain injury. Seizure prophylaxis may be considered. They may also require cardiovascular support in the form of IV fluids, inotropes, vasopressors and pulmonary dilators. Early fixation of the fracture is thought to avoid recurrent embolisation, but surgery is usually postponed until the patient is stable. There is evidence to suggest that steroids may reduce the incidence and severity of hypoxia. There is no indication for anticoagulation.

What is the physiological mode by which fat embolism syndrome occurs?

There are two main theories. The first is the mechanical theory that suggests that fat droplets from the bone marrow enter intermedullary vessels and move through the pulmonary circulation into the systemic circulation via arterio-venous or intracardiac

shunts. Once in the arterial system they become emboli. This results in local ischaemia and organ dysfunction. The second theory is the biochemical theory, which suggests that free fatty acids from the endothelial layer cause endothelial disruption resulting in a widespread inflammatory response which promotes agglutination of microemboli into large fat globules which increases the risk of mechanical obstruction. The inflammatory response can also activate the clotting cascade which can result in a prothrombotic state.

Further Reading

Barker RC Marval P et al. Venous thromboembolism: Risks and prevention *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11 (1): 18–23.

Jaff MR et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: A scientific statement from the American Heart Association. *Circulation*. 2011; 123 (16): 1788–830.

Luff D. et al. Fat embolism syndrome *British Journal of Anaesthesia Education*. 21 (9): 322–328.

National Institute of Clinical Excellence
Clinical Guideline 46. Venous Thromboembolism: Reducing the Risk of Venous Thromboembolism in Inpatients Undergoing Surgery. National Institute for Health and Clinical Excellence, 2007 www.nice.org.uk/guidance/CG92.

National Institute of Clinical Excellence
Guideline 158 Venous thromboembolic diseases: Diagnosis, management and thrombophilia testing www.nice.org.uk/guidance/ng158.

Hepatic Medicine

1.6.1 Hepatic Failure – Matt Thomas

This topic is presented as it might be encountered in the clinical science SOE, but could be met in the clinical section as a case-based discussion.

What are the major functions of the liver?

The liver is the largest gland in the body and many of its most important functions are metabolic, but there is also a significant immune function and an essential role in coagulation.

Tell me more about the metabolic role of the liver.

The liver has a role in almost all areas of metabolism, including carbohydrates, fats, proteins and urea, bilirubin and vitamins, not to mention drug and hormone metabolism. Regarding carbohydrates, the liver has a key part in glucose homeostasis; it stores glucose as glycogen and is the major site of gluconeogenesis from amino acids, lactate, pyruvate, and glycerol. The liver is the site of much protein synthesis, including albumin, clotting factors II, VII, IX and X, acute phase proteins, complement and cytokines, for example thrombopoietin. It is also the major site of urea synthesis from amino acids and ammonia. The liver synthesises and excretes bilirubin from haem breakdown products. The formation of bile is essential for elimination of lipid-soluble toxins and cholesterol and for the digestion and absorption of lipids and lipid-soluble vitamins. Some vitamins are stored in the liver, for example A, D, E and B12, while others, for example vitamin D, require the liver for their effect. Finally, the liver is the site of phase 1 and 2 metabolism of drugs and other toxins.

Not only does the liver protect the systemic circulation from the onslaught of material from the gut but it also has effects on systemic innate and adaptive immunity. Kupffer cells are modified macrophages within hepatic sinusoids that remove endotoxin and other antigens and pathogens derived from the portal circulation. Hepatic dendritic cells are potent phagocytes and cytokine releasers when stimulated. The liver synthesises complement and IgA is taken up and released into the biliary tree. Balancing the activation of the immune system, the liver has an important role in the development of immune tolerance to self.

And what about coagulation?

Well, the liver synthesises both pro- and anticoagulant proteins, and is a source of thrombopoietin, a stimulator of platelet synthesis. The pro-coagulants produced are factors II (pro- thrombin), VII, IX and X. Factors VII, IX and X are crucial in the

generation of thrombin from prothrombin when bound by tissue factor in the initiation of clotting. The liver also makes proteins C and S, which are part of the natural anticoagulation pathways. Synthesis of all these proteins is vitamin K dependent.

How do you define liver failure? What are the consequences?

Be succinct with your definitions. Refer to your previous answer about the functions of the liver to give the open-ended question about consequences a structure.

Liver failure may be chronic, acute-on-chronic or acute.

Acute liver failure is a syndrome of jaundice, encephalopathy and coagulopathy that occurs in individuals with no pre-existing liver disease. It is usually further divided into fulminant (or hyper-acute), acute and sub-acute liver failure according to the time between the onset of jaundice and the onset of encephalopathy, with intervals of 1 week, 2 to 4 weeks and 5 to 26 weeks respectively. This classification has some aetiological and prognostic relevance. As a rule of thumb hyperacute failure is associated with worse hepatic and extra-hepatic organ dysfunction but better transplant free survival, though age and underlying cause are important.

Chronic liver failure does not have a widely accepted definition, but is most often used to refer to signs of impaired hepatic function with or without background cirrhosis. A number of scoring systems using clinical and/or laboratory data may be used to classify chronic liver disease of which the Child-Pugh score and the Model for End-Stage Liver Disease (MELD) are most common.

Acute-on-chronic failure occurs when there is abrupt and progressive jaundice, coagulopathy and encephalopathy on a background of chronic liver disease. Extra-hepatic organ failure is common and in some definitions, is required for the syndrome to be diagnosed.

What kinds of data are used for the scoring systems you mention?

The Child-Pugh score uses bilirubin, albumin, INR, the extent of ascites and the presence of encephalopathy. The MELD score uses bilirubin, creatinine and INR.

Thank you. Now return to the consequences of liver failure.

Liver failure is the loss of its major metabolic and immune functions. Hypoglycaemia is common as glucose stores are depleted and gluconeogenesis impaired. Protein synthesis is markedly reduced with falls in serum albumin, acute phase proteins and clotting factors, the latter contributing to the coagulopathy of liver failure (as does a reduced absorption of vitamin K). Urea synthesis is impaired and systemic ammonia levels rise; this is thought to contribute to the development of encephalopathy, although the pathological basis is not fully understood. Jaundice develops as unconjugated bilirubin accumulates because of impaired glucuronidation. Immunity is impaired, particularly innate immunity, leading to increased bacterial and fungal infections.

So, what are the clinical consequences?

There is an intense systemic inflammatory response to hepatocyte damage and death which can look like severe sepsis. Clinical features that accompany acute liver failure are: hypoxaemia, hypotension with a high cardiac output, low SVR state and abnormal

cerebral, splanchnic and renal blood flows and consequently organ function, anaemia and thrombocytopenia, acute renal failure, acidosis and hyperlactataemia. There may also be worsening of the complications of chronic liver disease in acute-on-chronic failure. Infection, cerebral oedema and multiorgan failure are the leading causes of death in acute liver failure, which with the exception of paracetamol induced failure, carries a very high mortality in the absence of transplantation.

Can you expand on the complications of chronic liver disease?

These are essentially the complications of cirrhosis. Those of particular interest are portal hypertension and its sequelae such as varices and splenomegaly, ascites, hepatorenal syndrome and hepatopulmonary syndrome. There may also be additional complications associated with particular causes of cirrhosis, for example, cardiomyopathy associated with alcoholic liver disease. Patients are frequently malnourished and deconditioned.

Let's move back to acute liver failure. What are the common causes in the UK?

The aetiology of acute liver failure does vary according to type, but considering all together the most common cause in the UK is drug-induced liver injury, commonly paracetamol, followed by viral infections. In up to one in five cases no cause is found. Acute-on-chronic liver failure is commonly precipitated by sepsis, variceal bleeding, or hypotension and hypovolaemia from any cause, and by drugs including alcohol, diuretics and sedatives.

Which viruses and drugs are involved?

The viruses are hepatitis viruses A to E, and others such as cytomegalovirus (CMV), Epstein-Barr virus (EBV) and varicella zoster virus (VZV), though patients may be seronegative. One common drug precipitant, especially in younger patients, is ecstasy (MDMA) but antiepileptic drugs like phenytoin and carbamazepine, statins, chemotherapy and antimicrobials are also implicated.

And what causes chronic liver disease?

Chronic liver disease is an increasing cause of mortality in the UK and is now the fifth most common cause of death. The commonest cause in the UK is alcohol. Other causes are nonalcoholic steatohepatitis (often associated with diabetes and obesity), chronic viral infections, metabolic and autoimmune diseases and drug reactions.

Now I would like to consider a couple of clinical scenarios. First, take me through your assessment and management of a 25-year-old man presenting with 3 days of jaundice and 1 day of marked confusion and epistaxis. He has a history of severe depression and IV drug use and recently lost his job.

A clear and sensible structure to your answer is a good way of impressing the examiner while ensuring you do not forget important details. In this case there are two parts to the answer: assessment and management. Answer each in turn.

The history is suggestive of acute liver failure, although a similar picture could arise from severe sepsis or endocarditis and these may be difficult to distinguish.

This patient may be seriously unwell, so I would begin my assessment with a rapid review of vital systems using an ABCDE approach to identify immediately life-threatening problems. Then I would take a history from the patient if possible and friends or relatives if not, paying particular attention to potential causes of liver failure, in this case paracetamol overdose or other drug and alcohol use, and exposure to hepatitis B and C. I would examine the patient further, looking in particular for signs of chronic liver disease.

He does not have any signs of chronic liver disease, and looks as you suggest, seriously unwell.

Next I would order investigations to confirm my provisional diagnosis of acute liver failure, ascertain the aetiology and assess the severity, and to try to exclude alternatives. In this case I'd like a full blood count, clotting screen and fibrinogen, urea and electrolytes, liver function tests, calcium, magnesium and phosphate, paracetamol levels, arterial blood gases with lactate, an ECG and chest X-ray, and an ultrasound of the liver to assess size and patency of the portal vein and common bile duct. I would consider a triple-phase CT of the liver especially if ultrasound is inconclusive. Blood should also be taken for culture and tested for hepatitis viruses A, B and C and HIV, and urine tested for blood, protein and a drug screen. While waiting for results I would give oxygen, resuscitate with IV fluid, and start N-acetylcysteine (NAC) and broad-spectrum antibiotics.

I am interested in your use of N-acetylcysteine and antibiotics. Can you explain yourself?

Paracetamol overdose is the most common cause of hyperacute liver failure and is readily treated with NAC, which has a wide therapeutic range and may be of benefit in late presentation and some would say even other forms of liver failure. I would give antibiotics because sepsis is difficult to exclude clinically as a cause or consequence of this patient's condition and mortality increases with each hour delay in administration, and also because prophylactic antibiotics are indicated in severe liver failure.

Which antibiotics do you suggest?

I would use tazocin, for its gram-positive and especially gram-negative cover and fluconazole as up to 30% of infections in acute hepatic failure are fungal. Although they may not improve mortality, use can reduce incidence and severity of sepsis and encephalopathy which might facilitate transplantation. The possibility of viral reactivation – something like CMV – should always be considered.

I see. Now, it turns out that his paracetamol level is high and there is no history of recent IV or other drug use. He has a GCS of 9, and a bilirubin of 250, INR of 10, creatinine 450 $\mu\text{mol/L}$, and pH 7.2. Please continue.

The most likely diagnosis is that of paracetamol-induced fulminant hepatic failure, and this man has evidence of multiorgan failure. I would manage this patient in an intensive

care unit. Management includes both treatment and supportive care. Regarding the latter, I would intubate and ventilate to protect his airway and control carbon dioxide as he has a very high risk of developing cerebral oedema and raised intracranial pressure. Fluid resuscitation is required and I would use 0.9% saline and albumin, with vasopressors as necessary to keep mean arterial pressure above 65 mmHg in the absence of ICP and jugular saturation monitoring.

Sedation is necessary and agents like propofol and alfentanil are appropriate. Other supportive care, such as early nutrition and stress ulcer prophylaxis should be started and antibiotics continued. Treatment with NAC should also be continued and the patient referred to a liver transplant unit.

Do you know the criteria for transplant in this case?

The King's College Hospital criteria for paracetamol overdose is either a pH of less than 7.25, or all of the following three signs, a prothrombin time greater than 100 seconds, creatinine greater than 300 $\mu\text{mol/L}$ and grade 3 or 4 encephalopathy. These are usually applied after resuscitation, and the local transplant unit will advise on whether or not transfer is appropriate.

Would you put in a central and arterial line and would you correct the INR to do so?

Yes I would insert a central and arterial line. However, the INR is a key prognostic tool and should not be artificially corrected even to gain central venous access. Platelets may be given if low.

What are your thoughts on blood purification in acute liver failure?

Well, a liver equivalent of dialysis has long been sought and some systems are in use in specialist ICUs. In a general ICU either renal replacement or plasma exchange could be provided but the evidence to support routine use is not compelling especially if not a bridge to transplant. I would not start either for this indication without discussion and preferably transfer to a specialist unit.

Right. Now let's turn to a different patient presenting for elective hemicolectomy for carcinoma. They have alcoholic liver disease with ascites and are still drinking. What are the potential problems?

The presence of ascites signals significant disease as the liver normally has a large functional reserve. As a result, perioperative problems are more likely, in particular the risk of acute-on-chronic liver failure or hepatorenal syndrome. Other problems may be considered system by system. The liver metabolises most drugs used in anaesthesia, and drug effects are likely to be pronounced and elimination delayed. Ascites will reform postoperatively and must be accounted for in fluid balance calculations as significant hypovolaemia and electrolyte disturbance may result. It can also compromise respiratory function. Patients are frequently malnourished and need intensive nutritional support both before and after surgery. From a cardiovascular viewpoint this patient may have a

cardiomyopathy, or show the low blood pressure typical of cirrhotics. Hypotension and low cardiac output may compromise liver or renal function. They may be anaemic, thrombocytopenic or coagulopathic, which may lead to problems with bleeding and with regional anaesthesia. In chronic liver disease pulmonary shunts develop and ascites compromises respiratory function, and many of these patients are also smokers. This makes pulmonary complications like hypoxia and pneumonia more likely.

Yes, and can you think of anything else?

As mentioned, patients with liver disease are sensitive to the effects of opiates and anaesthetics and there is a risk of encephalopathy. There is an increased risk of infections such as peritonitis, pneumonia or wound infections, so antibiotic prophylaxis must be given. There may be electrolyte problems associated with drugs like diuretics used for ascites. And the effects of alcohol withdrawal must never be forgotten and benzodiazepines and an alcohol withdrawal score should be used.

So how would you optimise this patient for surgery?

In the first instance I would want to make a full assessment taking a history and examining the patient and getting a FBC, clotting screen, U&Es and LFTs, blood gases and ECG done at a minimum, with a group and save or cross-match according to the starting haemoglobin. An echo would be useful. From these the Child-Pugh score can be calculated which will give a rough indication of the risks associated with surgery so that the patient may be fully informed. Preoperative assessment would be best done well before surgery to allow any problems to be sorted out beforehand. The opinion of a hepatologist and any other relevant specialist should be sought to see if there was anything that could be done to improve the patient's medical condition. Advice on stopping drinking and the offer of referral to an alcohol liaison service to support this is also important.

Briefly tell me how would you approach the anaesthetic.

Very carefully! This patient is at high risk of morbidity and mortality and should have consultant anaesthetic and surgical involvement and HDU care perioperatively. Despite that, an enhanced recovery pathway is a reasonable starting point assuming there have been no complications during anaesthesia and surgery. I would use arterial blood pressure monitoring in addition to basic AoA and temperature monitoring and avoid hypotension and hypovolaemia. A urinary catheter and hourly urine output monitoring is also essential as fluid management is challenging especially in the presence of ascites. Propofol is suitable for induction and agents with minimal hepatic metabolism and rapid offset are ideal for maintenance. Atracurium would be a reasonable choice for neuromuscular blockade with close monitoring. If rocuronium is used, also with close monitoring, then sugammadex can be considered if coagulopathy is not too severe. Postoperative analgesic technique will depend on several factors, including clotting problems, presence of respiratory disease and the size and position of the surgical incision. Paracetamol may be used as long as liver function is carefully monitored and doses of opioids should be carefully titrated to effect. Other postoperative care will focus on avoiding precipitants of acute-on-chronic liver failure.

Further Reading

Aziz R, Price J, Agarwal B. Management of acute liver failure in intensive care. *British Journal of Anaesthesia Education*. 2021; 21: 110–116.

Seshadri A, Appelbaum R, Carmichael SP, et al. Management of decompensated cirrhosis in the surgical ICU: An American Association for the Surgery of Trauma Critical Care Committee Clinical

Consensus Document. *Trauma Surgery and Acute Care Open* 2022; 7: e000936.

Starczewska M, Mon W, Shirley P. Anaesthesia in patients with liver disease. *Current Opinion in Anaesthesiology* 2017; 30: 392–398.

Vaja R, McNicol L, Sisley I. Anaesthesia for patients with liver disease. *British Journal of Anaesthesia Education*. 2010; 10: 15–19.

1.6.2 Jaundice – Dana L Kelly and Andrew Weir

This topic is likely to be encountered in the basic science structured oral exam but could also form part of a long case discussion in the clinical structured oral exam. The basic science should be quick to cover, leaving plenty of time for the structured oral exam to discuss causes and implications of jaundice. It is important to be structured as this topic can potentially cover a vast amount of medicine and clinical anaesthesia.

Could you please define the term jaundice?

Jaundice (or icterus) is the yellow discolouration of the skin, the conjunctival membranes overlying the sclera, and other mucous membranes caused by hyperbilirubinaemia. Jaundice is not noticeable clinically until the bilirubin concentration is over at least 35 $\mu\text{mol/L}$.

It is derived from the French for yellow – jaune.

Please describe the normal formation and metabolism of bilirubin.

Bilirubin is formed when haemoglobin is broken down in the reticulo-endothelial system. The polypeptides of the haemoglobin molecule are cleaved from haem, an iron-containing porphyrin derivative. The haem is then in turn catabolised to biliverdin, a green tetrapyrrolic bile pigment.

Bilirubin is created by the activity of biliverdin reductase on biliverdin. Bilirubin, when oxidised, reverts to become biliverdin once again. This cycle, in addition to the demonstration of the potent antioxidant activity of bilirubin, has led to the hypothesis that bilirubin's main physiologic role is as a cellular antioxidant.

Regarding the metabolism of bilirubin, lipid-soluble unconjugated bilirubin is bound to albumin and transported to the liver. Hepatic conjugation occurs, converting unconjugated bilirubin into water-soluble conjugated bilirubin.

The conjugated bilirubin is then excreted into bile and stored in the gallbladder. Some of the conjugated bilirubin remains in the large intestine and is metabolised by colonic bacteria to urobilinogen, which is further metabolised to stercobilinogen, and finally oxidised to stercobilin.

Some of the urobilinogen is reabsorbed and excreted in the urine along with an oxidised form, urobilin. Stercobilin and urobilin are the products responsible for the colouration of faeces and urine, respectively. The remaining urobilinogen is reabsorbed from the gut and undergoes entero-hepatic re-circulation.

Could you classify the causes of jaundice?

There are four potential causes of hyperbilirubinaemia; increased bilirubin production, impaired conjugation, congenital abnormalities of bilirubin transport, obstruction of bile drainage.

To look at these in turn:

- Increased production occurs with haemolysis. Free bilirubin concentrations rise, but rarely to very high levels (e.g., $<50 \mu\text{mol/L}$). This is because the liver has substantial reserve to handle the increased production.
- Impaired conjugation occurs for many reasons, e.g., hepatitis, cirrhosis, drug-related hepatic failure. Unconjugated bilirubin is raised and urinary urobilinogen may be raised, as the liver is unable to excrete it.

Congenital abnormalities of bilirubin transport are rare, except for Gilbert's syndrome. This affects 5–10% of the population, and results in occasional episodes of isolated unconjugated hyperbilirubinaemia.

Obstruction of bile drainage can occur due to both extrahepatic and intrahepatic causes, and will result in a rise in conjugated bilirubin levels. Extrahepatic causes for biliary outflow obstruction include gallstones or pancreatic malignancy. Intrahepatic causes include infective and alcoholic hepatitis, liver cirrhosis, primary biliary cirrhosis and primary sclerosing cholangitis. Cholestasis can occur in pregnancy or can be caused by certain drugs, e.g., contraceptives, neuroleptic agents or steroids. Itching is common, and classically the patient reports dark urine and pale stools, due to the urinary excretion of conjugated bilirubin.

Importantly, a single condition could be responsible for more than one mechanism occurring, e.g., hepatocellular damage. For this reason I believe it is more useful to classify jaundice using the above physiologically based system rather than the classic system of pre-hepatic, intrahepatic and post-hepatic jaundice.

What are the implications of a high bilirubin for an anaesthetist?

Clearly the specific implications of jaundice in the perioperative period are linked to the underlying aetiology. Establishing the cause of jaundice is extremely important because of specific accompanying morbidity depending on the underlying diagnosis, e.g., extreme anaemia in haemolysis, or cardiomyopathy in alcoholic cirrhosis.

It is also important to establish if the patient has an isolated hyperbilirubinaemia, or if the raised bilirubin represents a global impairment of hepatic function. In particular, the coagulation function should be considered, as the liver is responsible for the synthesis of the majority of the protein clotting factors. Coagulation can be significantly impaired, particularly with an intrahepatic cause for jaundice. This clearly has implications for surgery, but also regarding the application of regional or neuraxial techniques. The presence of other significant sequelae of chronic liver disease should also be looked for. These include portal hypertension and hepatopulmonary syndrome.

It is important to establish if patients with jaundice have an active infective hepatitis. Not only has anaesthesia in the acute phase of hepatitis been shown to worsen long-term hepatic function, there are clearly issues related to infection control and protection of theatre staff from the transmission of blood-borne infections.

The action of anaesthetic drugs can be significantly affected in patients with jaundice. This can be due to hepatocellular damage leading to impaired ability to eliminate drugs

using normal mechanisms (e.g., cytochrome P450 enzymes being converted to inactive cytochrome P420), or due to reduced liver protein synthesis leading to alterations in drug pharmacokinetics.

Anaesthesia in the presence of severe liver dysfunction can predispose to the development of hepatorenal syndrome. This is a life-threatening condition that consists of a rapid deterioration in renal function in individuals with cirrhosis of any cause. Deteriorating liver function due to acute stress (such as surgery and anaesthesia) can result in acute renal failure occurring in the immediate postoperative period. The underlying cause remains unclear, although the risk seems particularly high if serum bilirubin concentrations exceed 180 $\mu\text{mol/L}$.

Two types of hepatorenal syndrome have been identified. Type 1 hepatorenal syndrome results in a rapidly progressive decline in kidney function, with a very high associated mortality (>50%). Type 2 hepatorenal syndrome is associated with development of ascites that is resistant to management with diuretics. Management recommendations include maintaining intravascular volume with large quantities of fluid, usually albumin, and administration of mannitol to increase urine output.

High levels of bilirubin can directly depress myocardial conduction leading to significant conduction delay and resultant bradycardia.

Neurological function can be significantly impaired, a situation which can be worsened by general anaesthesia. Hepatic encephalopathy is caused by accumulation in the bloodstream of toxic substances that are normally removed by the liver. It is managed by suppressing the production of the toxic substances in the intestine. This is most commonly achieved with high doses of laxatives. Hyperbilirubinaemia is not normally responsible for a reduced conscious level except in cases where there is a defective blood-brain barrier such as in neonates or in central nervous system infection.

Please consider the following case:

A 34-year-old man has presented for a day-case knee arthroscopy. He plays rugby regularly and has no significant medical history. He takes no regular medications. The extremely keen orthopaedic FY1 has taken some preoperative blood tests that show the following: Hb 134 (130–160 g/L), WCC 5.5 (4–11 $\times 10^9/\text{L}$), platelets 350 (150–400 $\times 10^9/\text{L}$), MCV 92 (76–96 fL); Na^+ 140 (135–145 mmol/L), K^+ 4.2 (3.5–5 mmol/L), urea 5 (2.5–6.7 mmol/L), Cr 75 (70–150 $\mu\text{mol/L}$); bilirubin 38 (3–17 $\mu\text{mol/L}$), ALT 14 (3–35 IU/L), AST 20 (3–35 IU/L), alkaline phosphatase 42 (30–300 IU/L).

What is the likely diagnosis?

These tests show an isolated hyperbilirubinaemia. I would ideally like to see a conjugated/unconjugated differential, but in view of the clinical history, the results suggest the patient has Gilbert's syndrome.

Tell me more about Gilbert's syndrome.

Gilbert's syndrome is the most common hereditary cause of increased bilirubin. It is found in up to 5–10% of the population. It has an autosomal recessive pattern of inheritance. The main symptom is otherwise harmless jaundice, caused by elevated levels

of unconjugated bilirubin in the bloodstream. This is related to the reduced activity of the enzyme glucuronyltransferase, which conjugates bilirubin and some other lipophilic molecules. Conjugation renders the bilirubin water-soluble, after which it is excreted in bile into the duodenum. Gilbert's syndrome is caused by a 70–80% reduction in the glucuronidation activity of the enzyme uridine–diphosphate–glucuronosyltransferase isoform 1A1.

In patients with Gilbert's syndrome, mild jaundice may appear under conditions of exertion, stress, fasting and infections, but the condition is otherwise usually asymptomatic. The unconjugated bilirubin in Gilbert's syndrome rarely exceeds 50 $\mu\text{mol/L}$.

Are there any implications of Gilbert's syndrome for the anaesthetist?

It is important to be certain that other more serious causes of hyperbilirubinaemia have been considered and excluded.

It is also important to be aware that patients with Gilbert's syndrome may have altered capacity to metabolise certain drugs. Of particular note is the association of Gilbert's syndrome and increased sensitivity to paracetamol toxicity.

What would be your concerns if a patient developed jaundice postoperatively?

I would take a detailed history and perform a thorough examination. A new-onset jaundice immediately postoperatively would raise concerns of haemolysis related to an adverse drug reaction or blood-product transfusion reaction. It would be important to conduct an analysis of the anaesthetic technique applied, particularly related to the use of volatile anaesthetics. Classically halothane is known to cause hepatotoxicity, although this agent is now used rarely in the United Kingdom.

Hepatitis of unknown aetiology has been reported following the use of isoflurane and sevoflurane. I would also want to ensure there was not hepatic damage as a result of severe intraoperative hypoxia or hypotension, which should be evident on the anaesthetic record. I would consider infection, as sepsis can present as a derangement of hepatic function. Depending on the operation, I would want to exclude surgically induced iatrogenic biliary obstruction. I would also consider a pre-existing medical or surgical condition that may have not been apparent preoperatively.

Can you tell me more about halothane hepatotoxicity?

This is mostly of historical interest now, but may come up in a structured oral examination if you are doing very well!

Two types of hepatotoxicity are associated with halothane administration. These are termed type 1 (mild) and type 2 (fulminant).

Type 1 hepatotoxicity is common affecting up to 25–30% of individuals who receive halothane. It is benign and self-limiting. The diagnosis is made by mild transient increases in serum transaminase and occasionally by altered postoperative drug metabolism. Type 1 hepatotoxicity is not characterised by jaundice or clinically evident hepatocellular disease. Type 1 probably results from reductive biotransformation of halothane rather than the normal oxidative pathway. It does not occur following administration of other volatile anaesthetics.

Type 2 hepatotoxicity, or halothane hepatitis, is associated with massive centrilobular liver necrosis that leads to fulminant liver failure. It is a very serious condition characterised by marked jaundice and grossly elevated serum transaminase levels. There is an associated mortality of >50%. It appears to be immune-mediated. Halothane metabolites bind liver proteins and, in genetically predisposed individuals, antibodies are formed to this metabolite–protein complex. Volatile anaesthetics other than halothane also have the potential to cause type 2 hepatotoxicity. This risk is directly related to the relative degree of their oxidative metabolism. Approximately 20% of halothane is oxidatively metabolised, compared to 0.2% of isoflurane. Hence halothane carries a higher risk of hepatotoxicity.

Further Reading

- Mastoraki A, Karatzis E, Mastoraki S, et al. Postoperative jaundice after cardiac surgery. *Hepatobiliary Pancreatic Disease International*. 2007; 6: 383–387.
- Vaja R, McNicol L, Sisley I. Anaesthesia for patients with liver disease. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2010; 10(1): 15–19.
- Walton B, Simpson BR, Strunin L, et al. Unexplained hepatitis following halothane. *British Medical Journal* 1976; 1: 1171–1176.
- Weitz J, Kienle P, Bohrer H, et al. Fatal hepatic necrosis after isoflurane anaesthesia. *Anaesthesia*. 1997; 52: 892–895.

Neurological and Muscular

1.7.1 Myasthenia Gravis and Muscle Diseases – Helen L Jewitt and Stephanie Wallis

You are scheduled to anaesthetise a 37-year-old woman with myasthenia gravis for a thymectomy. What are the anaesthetic implications of this condition?

Although relatively uncommon in routine practice, myasthenia gravis is one of a group of muscle diseases which are a beloved exam topic. This is because for each there are a number of specific perioperative issues. Your knowledge is explored through discussion of these issues. A useful way to introduce your answer is with an opening statement explaining the basis of the disorder and the main clinical features.

Myasthenia gravis is an autoimmune disease affecting the neuromuscular junction. The patient produces IgG antibodies against their own acetylcholine receptors. There is a reduction in the number of active receptors at the neuromuscular junction. The clinical picture is of weakness of the ocular, bulbar and proximal limb muscles which is exacerbated by exercise. In general, young women and older men are affected and there is an association with other autoimmune conditions and abnormalities of the thymus.

Describe your preoperative assessment of this patient.

In my preoperative assessment I wish to gain the information needed for a routine anaesthetic assessment in addition to specific details regarding the patient's condition. With respect to the myasthenia, my aims are to establish the duration and severity of the patient's symptoms and the regime of medication required to produce symptomatic improvement. It is vital to establish the degree of bulbar involvement as this has implications on the risk of reflux during general anaesthesia. The presence of significant respiratory involvement is also important as these patients are more likely to require ventilatory support postoperatively. Respiratory function tests may be appropriate to quantify the degree of baseline muscle impairment.

How would you optimise the patient?

Those with poorly controlled symptoms should have their pharmacological treatment optimised before surgery. This includes anticholinesterases, steroids and other immunosuppressant agents. Very rarely a severely affected patient will require preoperative plasmapheresis. The patient's normal anticholinesterase dose should be continued up

until the time of surgery. Sedative premedication should be avoided if possible as it can worsen respiratory failure. However, the judicious use of benzodiazepines is appropriate in anxious patients. Patients with significant respiratory involvement are likely to have a poor cough and will benefit from preoperative physiotherapy. A critical care bed should be available postoperatively for patients with severe or unstable disease.

Describe the conduct of your anaesthetic.

Thymectomy is carried out either via a median sternotomy or a minimally invasive thoracoscopic technique. In either case general anaesthesia with endotracheal intubation is required.

If the airway is compromised as a consequence of the thymus mass, a gaseous induction may be considered to maintain spontaneous breathing. Patients with myasthenia have a destruction of acetylcholine receptors and are therefore very sensitive to the effects of non-depolarising neuromuscular blocking drugs. These are ideally avoided if possible or used at a much reduced dose, for example one tenth of the normal intubating dose. If avoidance of neuromuscular blockade is not possible, the combination of rocuronium and sugammadex is advocated. Incremental doses of rocuronium may be needed and quantitative neuromuscular monitoring facilitates this titration.

A rapid sequence induction can be performed if indicated by patient factors; however, these patients are relatively resistant to the effects of suxamethonium. This is because reduced numbers of active receptors lead to a resistance to depolarisation. There is evidence to suggest that a dose of 1.5–2 mg/kg produces reliable intubating conditions.

Maintenance of anaesthesia with volatile or a total intravenous technique is appropriate.

It is mandatory to confirm full recovery of neuromuscular function prior to emergence. If possible the use of reversal with neostigmine is avoided as this may provoke a cholinergic crisis. Sugammadex does not present this risk as its mechanism of action is via encapsulation.

The approach used for the surgery will be important in planning a postoperative analgesic regime. As in the preoperative period any drugs which can produce respiratory depression should be used cautiously.

What factors can be used to predict the likelihood of this patient requiring postoperative respiratory support?

There are several factors associated with an increased incidence of respiratory failure postoperatively. These include patient factors such as a BMI of more than 29 and coexisting COPD, disease factors such as a disease duration of more than two years, bulbar or respiratory symptoms, a previous history of myasthenic crises, a daily pyridostigmine dose of more than 750 mg and a preoperative vital capacity of less than 2.9 litres. Surgical factors such as major blood loss and lung resection are also relevant.

What are the other postoperative concerns?

These patients need effective analgesia, chest physiotherapy and the early re-establishment of their normal medication.

What do you understand by the terms myasthenic and cholinergic crisis and how would you differentiate between the two?

A myasthenic crisis results from a relative lack of anticholinesterase. It may be precipitated by missed doses of medication, surgery or intercurrent illness. It is characterised by sudden, rapidly worsening weakness.

A cholinergic crisis is provoked by administration of an anticholinesterase. Acetylcholine levels in the plasma are increased leading to weakness with prominent muscarinic effects. These include bradycardia, hypotension, bronchospasm, increased salivation and sweating, pupillary constriction, abdominal pain and diarrhoea.

A dose of short-acting anticholinesterase such as edrophonium will improve the features of a myasthenic crisis but exacerbate a cholinergic crisis.

What is myasthenic syndrome?

Myasthenic syndrome is also known as Lambert-Eaton Syndrome and is a paraneoplastic condition. It is most often associated with small cell carcinoma of the lung. It is characterised by proximal muscle weakness more commonly affecting the lower limbs. This weakness is seen to improve with activity in contrast to that seen in myasthenia gravis. There can also be autonomic effects in the form of hypotension, urinary hesitancy and constipation.

The syndrome is thought to result from antibodies which are directed against the calcium channels in the presynaptic membrane of the neuromuscular junction. The inactivation of these channels leads to a decreased presynaptic release of acetylcholine in response to an action potential. Anticholinesterases do not improve the clinical features of the syndrome.

Individuals with myasthenic syndrome are sensitive to both depolarising and non-depolarising neuromuscular blocking agents.

A 40-year-old man presents for arthroscopy of the knee. He has a diagnosis of dystrophia myotonica. What is myotonia and what do you understand about this disorder?

Myotonia is a term to describe the failure of muscle to relax following contraction. In dystrophia myotonica there is an abnormality of sodium conductance within the muscle fibre leading to prolonged contraction.

What are the clinical features?

Most books have a long list of clinical features. Aim to organise your answer by systems, concentrating on those that have potential anaesthetic implications.

There are a characteristic group of clinical features. These include general features; frontal balding, cataracts and muscle wasting affecting the sternocleidomastoid and proximal limb muscles. There can be bulbar involvement with poor swallow, slurred speech and recurrent aspiration. Weakness of respiratory muscles can lead to poor clearance of secretions and a restrictive lung deficit. Long-standing hypoventilation can result in obstructive sleep apnoea and cor pulmonale. In the cardiovascular system the patient may have cardiomyopathy and conduction defects. Oesophageal motility and

gastric emptying may be affected. There is an association with diabetes and hypothyroidism.

What are the potential issues relating to anaesthesia in this patient?

The concerns relate to the patient's underlying cardiac and respiratory disease, their sensitivity to anaesthetic drugs and the risk of precipitating myotonia in the perioperative period. Appropriate investigations such as ECG, echocardiogram, arterial blood gases and lung function tests should be used preoperatively if there is a suspicion of cardiorespiratory problems.

Patients are acutely sensitive to sedative and hypnotic agents such that premedication should be avoided and induction agents used cautiously. Local or regional techniques should be used wherever possible. If general anaesthesia is used antacid premedication is recommended and intubation is likely to be required due to the risk of aspiration. Depolarising muscle relaxants are avoided due to a risk of precipitating myotonia. Invasive monitoring is used in cases with significant cardiomyopathy. Intraoperatively maintenance of normothermia is important as cold and shivering are recognised triggers for myotonia. Postoperatively a period of observation in a critical care setting is appropriate.

What is muscular dystrophy?

The muscular dystrophies are a group of inherited muscle diseases. They are characterised by gradual destruction of skeletal muscle. Importantly there is also commonly involvement of cardiac muscle. There is a spectrum of severity depending on the genetic mutation and the degree of muscle involvement.

The classification of these disorders is based on the pattern of inheritance which can be sex-linked, autosomal dominant or autosomal recessive. The most common form of muscular dystrophy is Duchenne's which has a sex-linked recessive inheritance and therefore mainly affects boys. It is the most severe form of the disease and presents with the onset of muscle weakness between the ages of three to five years. This weakness is progressive and most patients are confined to a wheelchair by their early teens. Life expectancy is approximately 25 years with death resulting from cardiac or respiratory failure.

Becker's muscular dystrophy has a similar inheritance pattern but a milder clinical picture. Autosomally inherited forms of the disease include facioscapulohumeral dystrophy which shows a dominant pattern and limb-girdle dystrophy which is recessive.

Although these syndromes are infrequently encountered in routine practice the type of surgery these patients present for includes scoliosis correction and treatment of contractures.

What are the clinical features?

There can be severe respiratory muscle weakness which may be exacerbated by a restrictive deficit due to kyphoscoliosis. Impaired clearance of secretions and recurrent chest infections are common.

Cardiomyopathy is a common finding and there is a significant risk of perioperative arrhythmias.

Exposure to suxamethonium can provoke rhabdomyolysis and significant hyperkalaemia due to potassium efflux from cells. Suxamethonium should therefore be avoided. There are concerns regarding a possible association between muscular dystrophy and a reaction similar to malignant hyperpyrexia on exposure to suxamethonium or volatile agents. This is based on case reports and has so far not been substantiated by prospective studies. Volatile agents are advocated by some for short cases but are generally avoided for lengthy procedures.

Further Reading

- Daum P, Smelt J, Ibrahim I. Perioperative management of myasthenia gravis *British Journal of Anaesthesia Education*. 2021; 21 (11) 414–419.
- Driessen J. Neuromuscular and mitochondrial disorders: What is relevant to the anaesthesiologist? *Current Opinion in Anaesthesiology*. 2008; 21(3) 350–355.
- Marsh S, Pittard A. Neuromuscular disorders and anaesthesia, part 2: Specific neuromuscular disorders *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11(4) 119–123.
- Marsh S, Ross N, Pittard A. Neuromuscular disorders and anaesthesia, part 1: Generic anaesthetic management *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11(4) 115–118.

Respiratory

1.8.1 Pneumothorax – Matthew P Morgan and Stephen Pearson

Describe the anatomy of the pleura.

The pleurae are a serous membrane that covers the lungs and mediastinum. It consists of two layers, the parietal pleura and the visceral pleura. The visceral pleura covers the lungs and mediastinal structures and the parietal pleura lines the thoracic cavity. Between these two layers is a potential space containing a small volume of serous fluid. This fluid has two main functions that are very important for breathing: firstly, to lubricate the layers so they can slide with minimal resistance as the lungs expand and relax; secondly, to provide a surface tension between the two layers, keeping the space between them closed and the underlying lung fully expanded. The parietal pleura neurovascular supply comes from the intercostal nerves and vessels, while the visceral pleural only has autonomic innervation and its blood supply comes from the bronchial vessels.

Why is the inter-pleural space so important, even in health?

This space is normally maintained at a sub-atmospheric pressure of between minus 0.5 and minus 1.5 kPa, depending upon the level on the chest that is measured. This negative pressure is due to the elastic recoil of the chest wall springing outwards and the elasticity of the lungs pulling inwards, factors which are also important in determining functional residual capacity. The regional differences in inter-pleural pressures from the top to the bottom of the lung are due to the effect of gravity on lung mass with lower regions being under less negative pressure than those at the apex. This in turn has important consequences when examining a pressure–volume compliance curve and explains why, under normal circumstances, the upright lung is ventilated greater at the bottom compared with the apex. It can be seen therefore that the pleura and the pleural space are essential for forming the changes in pressure which ultimately cause lung expansion.

A 20-year-old man who is normally fit and well, presents with sudden onset pleuritic chest pain and breathlessness with no history of trauma. A chest X-ray shows a 3 cm right-sided pneumothorax.

How is a pneumothorax classified and how will you manage this patient?

A pneumothorax occurs when air enters the pleural cavity. Pneumothoraces are classified as spontaneous or traumatic. Traumatic can be either as a result of direct or indirect trauma and spontaneously occurring pneumothoraces are further classified as being primary or secondary.

A secondary spontaneous pneumothorax is when there is underlying lung disease and a primary spontaneous pneumothorax is in an otherwise healthy patient without lung disease.

This patient does not have lung disease and no history of trauma; therefore he has a primary spontaneous pneumothorax.

The management of a pneumothorax depends on whether it is primary or secondary, the size of the pneumothorax (measured at the hilum), and degree of breathlessness.

This patient has a primary pneumothorax that is 3 cm and he has significant breathlessness. He would need an A to E assessment and initially given high flow oxygen. The current British Thoracic Society guidelines suggest that in this situation, needle aspiration should be attempted.

How would this be performed? What is the 'safe triangle'?

After gaining written informed consent and checking for contraindications, with full monitoring I would prepare my needle aspiration kit. I would prepare the skin and identify the 'safe triangle'. The boundaries of this triangle consists of the anterior border of the latissimus dorsi muscle, the lateral border of pectoralis major muscle, the 5th intercostal space and the apex of the axilla. Using the safe triangle reduces the risk of injury to the liver, spleen or heart.

After local anaesthetic infiltration of the skin and subcutaneous tissues, a 16- or 18-gauge cannula or needle aspiration kit should be inserted close to the superior border of the rib to avoid the neurovascular bundle. Up to 2500 ml of air should be aspirated and then a further chest X-ray should be taken. If the pneumothorax has been reduced to less than 1 cm with improvement in symptoms, the patient should be monitored and given high flow oxygen and no further intervention would be required.

How would your management differ if the patient was requiring a general anaesthetic with positive pressure ventilation?

Positive pressure ventilation is not only a risk factor for pneumothorax due to barotrauma but it has the potential to drastically worsen an existing pneumothorax. In normal circumstances, ventilation is achieved using the negative pressure created by expanding the thoracic cavity by contraction of the diaphragm and intercostal muscles. However, air is forced under pressure into the lungs during positive pressure ventilation. This can rapidly expand a small simple pneumothorax and potentially create a tension pneumothorax which is a life-threatening emergency. The defect in the pleura that caused the initial pneumothorax will act as a one-way valve, allowing air into the pleural cavity but not to escape. This will rapidly increase the size of the pneumothorax which will cause the lung to collapse and put pressure on the heart and great vessels causing haemodynamic instability and potentially cardiac arrest.

Therefore, even a small primary spontaneous pneumothorax will require an intercostal drain connected to an underwater drainage system before starting positive pressure ventilation. A small-bore chest drain is shown to be as effective as a large bore drain and is easier to insert and associated with less pain.

Nitrous oxide should be avoided in patients with a pneumothorax as this can also cause rapid expansion of the space due to its relatively high blood gas solubility coefficient compared to nitrogen.

What is an underwater drainage system, what are its components and how does it work?

An underwater drainage system is attached to an intercostal drain to ensure drainage of pleural contents whilst minimising the risk of air entrainment. There are two main types in use, a one-bottle system and a three-bottle system, each of which consists of two main elements. Firstly, a tube is attached to the intercostal drain and positioned around 3–5 cm below the surface of a water filled bottle. This tube should be wide enough to minimise resistance to flow.

Secondly, the bottle should be watertight, clear and have a volume of water above the entry tube equal to half a vital capacity breath to prevent air in-drawing during deep breathing. The whole arrangement should be placed 50–100 cm below the level of the chest to prevent fluid entrainment.

What is the advantage of the three-bottle system you referred to?

This allows pleural fluid and air to be drained separately and accurately measured. Using the one-bottle system for this purpose would gradually alter the mechanics of the system as fluid is drained into the same bottle, which is providing an underwater seal. The water level would gradually increase over time leading to increased resistance to air drainage.

You are asked to see a patient on the intensive care unit with acute respiratory distress syndrome on a background of chronic obstructive pulmonary disease. She has just had an internal jugular central line inserted using a high approach under real-time ultrasound guidance. The nursing staff report that her oxygen saturations have reduced and her peak inspiratory pressures increased since the insertion of the central line.

What are the likely causes of this deterioration?

This patient is clearly at high risk of traumatic pneumothorax both from direct causes, including the insertion of a central venous catheter, and from indirect causes such as barotrauma. A pneumothorax following central venous cannulation can occur if the parietal pleura is breached during needle insertion and is more common when cannulating the subclavian vein. Although a high approach does reduce the risk of a pneumothorax occurring, the pleura can extend 3–5 cm above the clavicle cranially, thus a direct traumatic pneumothorax is still possible.

National Institute for Health and Care Excellence guidance recommend ultrasound for central venous cannulation; however, using an out-of-plane approach can still result in a pleural injury. An indirect traumatic pneumothorax is normally associated with barotrauma leading to bronchial or pleural bleb rupture and is more commonly seen in those with pre-existing lung disease or patients requiring high peak positive inspiratory pressure as is seen in ARDS.

Positioning patients for central venous cannulation would involve using a Trendelenburg position which in itself would increase the peak inspiratory pressures required if volume-controlled ventilation is being used. This positioning alone therefore may have precipitated an indirect traumatic pneumothorax. Apart from a pneumothorax, we would also want to consider other potential causes including endobronchial intubation, lobar collapse, mucus plugging, pulmonary embolus or an equipment problem.

A plain chest X-ray is performed on this patient and reported as normal. Does this exclude a pneumothorax as the cause of the deterioration?

Although plain chest X-rays are a useful first line investigation, they are not 100% sensitive and often miss small anterior pneumothoraces especially in supine patients. In patients with ARDS even such small insults can be sufficient to cause life-threatening hypoxia and therefore other modes of investigation should be used when clinical suspicion is high. Both the adhesions between pleural layers in chronic lung disease and the reduced lung compliance in ARDS can lead to unusual presentations of pneumothorax. Adhesions, for example, allow localised or loculated air collections, whilst due to poor lung compliance, a tension pneumothorax in patients with ARDS may show no signs of mediastinum shift. A lateral decubitus chest X-ray could be used although this is technically difficult on the intensive care unit and its sensitivity is also poor. Point-of-care lung ultrasound is increasingly commonly used in critical care and has been shown to be sensitive for diagnosis of small pneumothoraces. Finally, the gold standard for diagnosis would be a thoracic computerised tomography scan although the logistics in carrying out such an investigation would be challenging.

An intercostal drain was inserted in this patient but despite placement for 48 hours, the underwater drain is continuing to bubble. What is the most likely cause and how should it be managed?

As this is the Final FRCA be prepared to think around topics and incorporate the anaesthetic subspecialties, such as thoracic anaesthesia, into your answer.

There appears to be a persistent communication between the bronchial tree and the pleural space. This is known as a bronchopleural fistula. This can be a major problem for patients on the intensive care unit due to the inability to apply positive end-expiratory pressure, loss of tidal volumes, persistent lung collapse and prolonged duration of mechanical ventilation. In general, these patients are treated conservatively by continuing intercostal drainage with high volume, low-pressure suction and attempting to minimise tidal volumes, PEEP, inspiratory times, peak pressures and respiratory rate. This will often lead to permissive hypercapnia being utilised. Ideally, rapid weaning to spontaneous ventilation is the ventilatory mode of choice although this is often not possible. Advanced ventilatory strategies are occasionally used including differential lung ventilation using a double lumen tube. Small leaks can be sealed via a bronchoscope whilst larger leaks need formal surgical intervention. Respiratory physician and/or thoracic surgery review is necessary.

Further Reading

MacDuff A, Arnold A, Harvey J. Management of spontaneous pneumothorax: British

Thoracic Society pleural disease guideline. *Thorax*. 2010; 65: ii18–ii31.

1.8.2 Chronic Obstructive Pulmonary Disease and Asthma – Matthew P Morgan and Andrew Weir

This important topic is most likely to appear as part of the SOE.

How would you assess a 70-year-old man with chronic obstructive pulmonary disease who presents for an emergency laparotomy?

This is a common situation that you will have often encountered. The examiner will want to see a well structured approach demonstrating your competence at dealing with this important scenario.

The term chronic obstructive pulmonary disease (COPD) describes a wide spectrum of illness severity encompassing the clinical entity of chronic bronchitis and the pathological process of emphysema. Although these are two distinct entities, they frequently coexist. Preoperatively I would focus on assessing disease severity and looking for related comorbidities such as smoking-related ischaemic heart disease. Disease severity can be assessed clinically or by using investigations. Whilst taking a focussed history I would classify the patient's exercise tolerance according to the Medical Research Council's Dyspnoea Scale where a rating of 0 would describe no restriction of activity and a rating of 4 would describe breathlessness whilst undressing. Alternatively, I may quantify the distance a patient is able to walk on the flat or on an incline. I would ask about recent admissions to hospital with exacerbations of COPD including any previous intensive care admissions. I would actively seek evidence of current infection and quantify the amount of sputum produced. Finally, I would note the man's regular oral and inhaled medications, including the use of steroids and home oxygen therapy.

Moving on from the history, a focussed clinical examination may show evidence of a barrel-shaped chest, nicotine staining, bronchospasm, active infection or cor pulmonale with an elevated jugular venous pressure and hepatomegaly. The patient's pattern of breathing, including the use of accessory muscles and posturing, may reveal dyspnoea at rest. In addition, the use of pursed lip breathing to provide a degree of positive end-expiratory pressure, therefore preventing premature airway collapse, may also be seen. The presence of a long expiratory phase would indicate long time constants of some alveoli with resultant air trapping. Finally, I would want to assess this patient's nutritional status, as cachexia often coexists with COPD, and can lead to an increased mortality.

What investigations may be useful?

Try to avoid the temptation to jump straight into a discussion of lung function tests, but be prepared to move on swiftly if the examiner accepts that you would look at common blood tests first.

A full blood count may reveal polycythaemia as evidence of chronic hypoxia and an elevated neutrophil count may suggest active inter-current infection. Arterial blood gas analysis may show evidence of hypoxia and compensated type 2 respiratory failure with a normal pH despite hypercarbia as a result of an elevated bicarbonate concentration. An electrocardiogram may show evidence of right ventricular hypertrophy, right axis deviation and right atrial enlargement.

Moving on to more specialist investigations, I would expect this patient's pulmonary function tests to show an obstructive defect with the FEV1/FVC ratio being less than 70%, thus indicating small and medium-sized airway obstruction. The FEV1 expressed as a percentage of predicted values can be used to grade severity, with a FEV1 less than 30% predicted representing very severe disease in the NICE 2010 guideline. If an emphysematous disease process is also present, the carbon monoxide transfer factor may be reduced. Although an

absolute value of FEV1 less than 1 litre is included in the British Thoracic Society guidelines for assessment for thoracic surgery, there is little evidence for its application in non-thoracic surgery. We can say, however, that an FEV1 of more than 0.8 litres is required for an adequate cough and therefore values less than this would indicate a high chance of postoperative respiratory morbidity and mortality. In addition to these basic lung function tests, this patient may show evidence of reversibility with bronchodilators or steroids.

If flow/volume loops were obtained, they would show the characteristic 'scooped-out' appearance to the initial expiratory phase (Figures 1.8.2.1, 1.8.2.2, 1.8.2.3). This is because

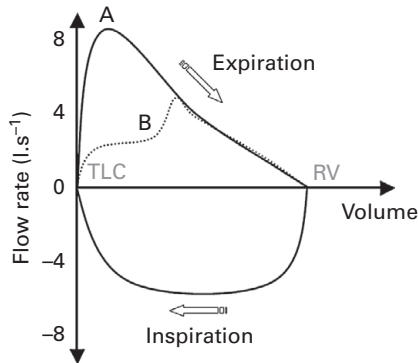


Figure 1.8.2.1 Normal flow-volume loop. TLC, total lung capacity; RV, resting volume.

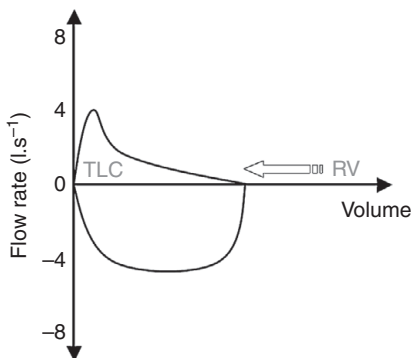


Figure 1.8.2.2 Obstructive disease flow-volume loop. TLC, total lung capacity; RV, resting volume.

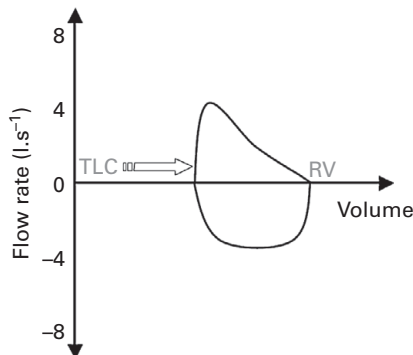


Figure 1.8.2.3 Restrictive disease flow-volume loop. TLC, total lung capacity; RV, resting volume.

maximal expiratory flow rates during the latter two thirds of an expiratory manoeuvre are largely effort independent but vary directly with the elastic recoil of the lung and inversely with the airway resistance upstream of the equal pressure point. Lung compliance may actually be increased if emphysematous changes predominate or may be reduced if chronic bronchitis is the main pathology.

An echocardiogram may show evidence of right ventricular failure as a result of chronic lung disease leading to elevated pulmonary artery pressures. Finally, cardiopulmonary exercise testing is now being increasingly used for risk stratification before major elective surgery. A VO_2 max of less than 15 ml/kg/min or an anaerobic threshold of less than 11 ml O_2 /kg/min has been shown to significantly increase postoperative mortality.

Your history and investigations suggest that this man has severe COPD. In addition, he has been shown to have an active pneumonia with an elevated neutrophil count and a mild pyrexia. What role should a regional technique play when considering postoperative analgesia in this patient?

There is no right answer to whether an epidural should or should not be inserted in this case. Instead, the examiner wants to see your thought process balancing the risks against the benefits of such an intervention.

As well as the usual contraindications to central neuraxial blockade such as clotting abnormalities and antiplatelet agents, we need to balance the risks of epidural abscess against the benefits that a regional technique could bring to this patient with severe chronic lung disease. Although the evidence at present is mixed, some studies have shown a reduction in mortality with epidural use in patients with COPD. Epidural abscess is a very rare complication of epidural use, occurring in around 1 in 200,000 episodes.

The third National Audit Project carried out by the Royal College of Anaesthetists was designed to look into the incidence and risk factors for such complications in the UK. The report found an incidence of epidural abscess following central neuraxial blockade of 1 in 24,000. The incidence of permanent harm was around 1 in 50,000. Identified risk factors included compromised immunity, anti-thrombotic drug therapy, traumatic procedure (multiple epidural attempts), source of infection, failure of aseptic technique and duration of catheter placement. In this case, unless the patient shows signs of severe systemic sepsis, I would proceed with epidural analgesia as I feel the benefits of an improved cough, improved respiratory function and reduced thromboembolic events outweigh the risks of epidural use, including that of epidural abscess.

What mode of ventilation would you use intraoperatively?

Again, there is no right answer to this question and you could justify your answer either way.

The two basic modes of ventilation used are volume and pressure directed ventilation. The former uses a constant flow generator to deliver a set tidal volume whilst the

latter uses a set pressure to deliver tidal volumes in accordance with the patient's total chest compliance. Both can be triggered either by time or by patient effort in the form of synchronised intermittent mandatory ventilation. The high peak pressures delivered when using constant flow generators have theoretical disadvantages for those patients with chronic lung disease. In addition, it is known that such patients have alveoli with a wide range of different time constants and therefore rates of inflation. Thus, using constant flow generators tends to over-expand some lung units whilst under-expanding others in accordance with Laplace's law. As this can result in a combination of barotrauma and atelectasis, it has been suggested that constant pressure ventilation may be of benefit.

Whichever mode of ventilation is used, it is important to provide sufficient time for exhalation by reducing the respiratory rate or I:E ratio to reduce the risk of gas-trapping. While studies addressing the use of positive end expiration pressure (PEEP) are equivocal, it is thought that the use of PEEP may help keep small airways open in late exhalation.

What can be done postoperatively to help reduce the problems associated with chronic lung disease?

Prior to extubation it is vitally important to make sure that any neuromuscular blockade has been reversed. Good analgesia with a regional technique will help with coughing and therefore sputum clearance as well as avoid the need for systemic opioids, which may result in type 2 respiratory failure. This man should be managed in a critical care environment, and regular physiotherapy is essential and may include the use of incentive spirometry and vital capacity breathing. Regular arterial blood gases and timely adjustment of the inspired oxygen concentration will help ensure that oxygenation is maintained whilst avoiding oxygen toxicity. Any supplemental oxygen used should be humidified to reduce drying of secretions.

What causes asthma?

A broad summary of asthma and its aetiological factors is required here but try not to get fixated on one or another particular cause.

Asthma is characterised by episodes of breathlessness, chest tightness and wheeze as a result of reversible airway narrowing. This in turn is due to a triad of smooth muscle spasm, mucosal oedema and increased secretions. It can generally be classified into atopic asthma, which occurs in early childhood in individuals with IgE hypersensitivity to common environmental allergens, and late onset asthma, which is not IgE related. The causes are multifactorial and controversial. There is evidence that environmental, genetic, infective and immunological factors all play a role in the pathogenesis of asthma.

Can you classify the pharmacological strategies used in the management of asthma?

These can be divided into three main groups: drugs causing bronchodilation, reducing mucosal oedema or modulating the immune response. Bronchodilators are generally sympathomimetic or anticholinergic. The sympathomimetic drugs act as β -adrenoreceptor agonists activating G proteins and therefore inducing bronchodilation. Examples include short-acting salbutamol and longer-acting salmeterol. Related drugs

such as ephedrine, adrenaline and the S+ stereoisomer of ketamine may be used in certain situations. Anticholinergics commonly used include inhaled ipratropium. Beware that neostigmine, by increasing the concentration of acetylcholine, can precipitate bronchospasm by antagonising this mechanism. Other agents which act by decreasing bronchospasm via a G protein mechanism include volatile anaesthetic agents and phosphodiesterase inhibitors such as theophylline. A reduction in mucosal oedema is achieved primarily by using inhaled and oral corticosteroids.

The pharmacological management of chronic asthma is set out in a stepwise fashion in the British Thoracic Society/SIGN guidelines.

Should positive end-expiratory pressure be used when anaesthetising an asthmatic for a laparotomy?

Positive end-expiratory pressure (PEEP) is generally used during major intra-abdominal surgery to help minimise basal atelectasis and hence reduce ventilation/perfusion mismatch. However, asthmatic patients tend to have high levels of gas trapping and develop high levels of intrinsic or auto PEEP. In theory, applying additional levels of extrinsic PEEP may cause hyperinflation of the chest, reduce venous return and increase peak inspiratory pressures. This in turn leads to a patient who is difficult to ventilate and may precipitate a pneumothorax. However, there is controversy in the use of PEEP in the intensive care management of acute asthma with some sources arguing that it can be of benefit.

Would you use non-steroidal anti-inflammatory drugs in people with asthma?

Although non-steroidal anti-inflammatory drugs are a very effective adjunct in multimodal analgesia, in around 10% of asthmatics they can precipitate bronchospasm. This is due to the biosynthesis of arachidonic acid derivatives being shifted towards leukotrienes and away from endoperoxides due to the inhibition of cyclo-oxygenase (COX) one and two. The groups most likely to be affected are those with atopic asthma, especially those with nasal polyps. Although a history of non-steroidal use is helpful in deciding whether they are safe to prescribe, different non-steroidal anti-inflammatory drugs have differing levels of COX inhibition and may therefore produce differing results. There are many other drugs used in anaesthesia that can precipitate bronchospasm including thiopentone, atracurium and suxamethonium.

Should steroid supplementation be given to an asthmatic on long-term inhaled fluticasone undergoing major surgery?

Classically, steroid supplements have been given to patients in order to avoid an Addisonian crisis precipitated by steroid withdrawal after long-term administration and a blunted stress response to surgery. Recent guidelines suggest that steroid supplementation should be given to adults taking more than 5 mg per day of prednisolone or equivalent (or 10–15 mg/m² in children) via whichever route of administration, including inhaled, as evidence has shown that suppression of the adrenal response to ACTH can occur even in recommended dose ranges. I would

therefore want to calculate the dose of inhaled steroid being used, and prescribe supplementation if indicated.

What is your approach to asthma in pregnancy?

Asthma affects around 1% of pregnancies with 10% of those requiring hospital admission either due to superimposed respiratory tract infection or worsened bronchospasm. Although asthma can improve, worsen or remain unchanged in pregnancy, these women should receive antenatal counselling and those with moderate to severe asthma should be encouraged to have early epidural analgesia. If operative interventions are required, a regional technique is the preferred method of choice. Regular medications should be continued during pregnancy and are overall thought to be safe. Salbutamol, for example, as a β -agonist acts as a tocolytic and therefore has been used to prevent early labour. However, those taking high-dose inhaled or oral steroids may show evidence of macrosomia and are more likely to develop gestational diabetes. Care should also be taken with the use of NSAIDs in the postpartum period.

Further Reading

Lumb A, Biercamp C. Chronic obstructive pulmonary disease and anaesthesia. *Continuing Education in Anaesthesia Critical Care and Pain*. 2014; 14(1): 1–5.

Health improvement Scotland. BTS/SIGN British Guideline for the management of asthma. *Scottish Intercollegiate Guidelines Network*. 2016; 158.

1.8.3 Pneumonia – Matthew P Morgan and Andrew Weir

This topic would be well suited to the SOEs. As this is such a common and important disease, you should be able to answer the questions confidently, accurately and with recent guidelines or evidence-based medicine at the forefront of your mind.

What are the different types of pneumonia?

A structured approach is vital to ensure that you do not forget any of the key points and a brief summary will instil confidence in the examiner's mind.

Pneumonia is an important and common clinical problem, and up to 10% of those attending hospital will require critical care admission. Although we have a range of antibiotics to treat the causative organisms, mortality from pneumonia remains around 17% in those admitted to critical care and as high as 30% for patients that develop severe sepsis. We can generally classify the disease into community-acquired, hospital-acquired and aspiration pneumonia. This classification gives us information not only about the likely pathogens, but also the pattern of lung involvement and likely prognosis. Community acquired pneumonia typically presents with the classic triad of fever, new consolidation on chest X-ray and clinical signs such as cough and dyspnoea. The most common causative organism found overall in community acquired pneumonia is the gram-positive aerobic coccus, *streptococcus pneumoniae*. Other so-called atypical pneumonias also feature including *mycoplasma*, *legionella* and *staphylococcus aureus*.

Hospital-acquired pneumonia is of particular importance for anaesthetists for two main reasons. Firstly, the vast majority of postoperative pneumonias are hospital-

acquired. Secondly, ventilator-associated pneumonia is also a type of hospital-acquired pneumonia. Here resistant organisms tend to be responsible including gram-negative species, staphylococcus aureus, methicillin resistant staphylococcus aureus (MRSA), pseudomonas and klebsiella.

Finally, an aspiration pneumonia is normally precipitated by an event leading to poor bulbar or oesophageal function. Causative organisms tend to be anaerobic and the prognosis is particularly poor. Despite this microbiological knowledge, most pneumonias are termed culture negative and no single causative organism is identified. This does not mean that a causative organism is not present or responsible but rather the influence of early antibiotic administration may affect its growth in culture. Patients should therefore be treated empirically according to clinical suspicion and their clinical condition and not simply on microbiological results. Where an organism is identified, therapy could thus be narrowed to a more specific agent.

What is the mechanism causing hypoxia in severe pneumonia?

Hypoxia in pneumonia results from a mismatch between oxygen supply and demand. Increased oxygen is required to sustain the systemic inflammatory response syndrome of tachycardia, pyrexia and high systemic inflammatory mediators. Supply is reduced due to ventilation/perfusion mismatch from bacteria and inflammatory cell parenchymal infiltration. Localised oedema also contributes which leads to overall reduced ventilation to affected lung areas. Hypoxic pulmonary vasoconstriction is less effective in these areas than during health due to the many inflammatory mediators released and hence there is an overall reduction in the ventilation/perfusion (VQ) ratio in these segments, also known as shunt.

What is ventilator-associated pneumonia?

Ventilator-associated pneumonia (VAP) is the most common hospital acquired infection in the intensive care unit, and has been shown to prolong mechanical ventilation and increase mortality by 30%. Although no formal diagnostic criteria exist, new or progressive pulmonary infiltrates with fever, leucocytosis, and purulent tracheobronchial secretions in patients ventilated for over 48 hours have been used to indicate the presence of VAP. Increasingly, evidence now suggests that directed or non-directed bronchioalveolar lavage (BAL) should be included in these criteria. The main pathogenesis is thought to be biofilm formation within endotracheal tubes and microaspiration of gastric contents and secretions, therefore explaining why the causative organisms are mainly gram-negative species as well as staphylococcus aureus and MRSA. The main risk factors identified which increase the risks of a VAP occurring include nursing patients in a fully supine position, prolonged ventilation, heavy sedation, inadequate cuff pressures and possibly the use of H₂ receptor antagonists, although this is controversial.

What are the correct cuff pressures, what cuffs are currently used and how do they contribute towards VAP?

Don't be put off by questions that are small print. The examiner is probing your knowledge and wants to see basic concepts applied to clinical practice.

The cuff of an endotracheal tube aims to minimise the passage of secretions from above to below the cuff. Obviously, greatly increasing the cuff pressure would form a

more secure seal in the short-term but these benefits must be balanced against the trauma that this would cause from mucosal ischaemia after long-term use. It is now thought that small folds appear in tracheal cuffs, which allow the passage of material from above to below therefore contributing towards the incidence of VAP. The use of high volume, low-pressure cuffs has helped balance the benefits of a secure seal against the risks of mucosal oedema and the use of new soft flexible silicon cuffs is becoming increasingly popular. A pressure of between 15 and 40 mmHg is considered acceptable in these high volume cuffs and protection can be optimised by using an endotracheal tube of correct size.

What can be done to minimise the risk of postoperative pneumonia in a patient with chronic lung disease undergoing a laparotomy?

This is a 'bread and butter' issue for anaesthetists and one that you should be able to talk at great length about. Unlike the previous small print question, performing poorly here would not be tolerated.

The two main important factors in preventing postoperative chest infection would include providing adequate analgesia and providing good quality respiratory care post-operatively. Regional analgesia, such as a thoracic epidural, will allow good quality pain relief to be achieved without giving systemic opioids which may result in hypoventilation, basal atelectasis and hence hospital-acquired pneumonia. Good regional analgesia should allow deep breathing and coughing to occur, again helping to prevent sputum retention and basal atelectasis.

Respiratory after-care should include regular chest physiotherapy, humidified inspired oxygen and the continuation of a patient's usual respiratory medications such as bronchodilators. For ventilated patients, one should follow the 'ventilator care bundle' as promoted by the Department of Health's 'Saving Lives' campaign. This bundle consists of four main elements: ensuring prophylactic anti-thrombotic measures are taken, ensuring gastric ulcer protection, maintaining the bed at a 30 degree head up tilt and providing regular sedation breaks where appropriate. The latter two of these components have been shown to reduce ventilator-associated pneumonia. An early tracheostomy in patients that are anticipated to undergo prolonged mechanical ventilation may reduce postoperative chest infection, although the TracMan study showed no mortality benefit.

You are asked to see a patient on the intensive care unit with community acquired pneumonia. He has been increasingly difficult to ventilate over the last 24 hours and now has an intermittent high-grade pyrexia. A plain chest radiograph shows evidence of a large pleural effusion. What are the possible causes of this effusion and how should it be managed?

The two main differential diagnoses in this patient would be an exudative pleural effusion and an empyema. Pleural effusions are defined as fluid between the visceral and parietal layers of pleura. For an effusion to be seen on plain chest radiography at

least 400 ml of fluid must be present and so smaller effusions may be demonstrated by CT or ultrasound. Effusions can be divided into transudates or exudates, where parapneumonic effusions tend to be exudate in type. Although it is normally stated that a transudate will have less than 30 g/L of protein, this is dependent upon the plasma albumin level. As this is often reduced in critically ill patients, a level of protein in pleural fluid greater than one half of the plasma level is accepted to be sufficient to term an effusion an exudate. Alternatively, this may be an empyema, a collection of bacteria and pus cells which normally has a pH less than 7.2. This would certainly account for the high pyrexias that the patient has been experiencing. If any effusion, regardless of type, is causing respiratory compromise, it should be drained by inserting an intercostal drain. If the effusion is exudative or an empyema, a large bore intercostal drain should be used using a surgical dissection method rather than the Seldinger approach used for smaller drains suited for the treatment of pneumothoraces. It is important that the fluid from the effusion is sent for microscopy and culture in order to direct anti-microbial therapy.

Further Reading

Gunasekera P, Gratrix A. Ventilator-associated pneumonia. *British Journal of Anaesthesia Education*. 2016; 16(6): 198–202.

Morgan AJ, Glossop AJ. Severe community-acquired pneumonia. *British Journal of Anaesthesia Education*. 2016; 16 (5): 167–172.

1.8.4 Lung Cancer and Interstitial Lung Disease – Alexander Midgley-Hunt

Lung cancers are rarely encountered as an anaesthetist in theatre; however, topics which examine rare conditions are often used to discriminate between the borderline candidate and those who are well prepared.

Lung Cancer

What pulmonary malignancies are you aware of?

Lung cancer can either be a primary tumour or a secondary tumour. Primary tumours are generally associated with smoking and are divided into 2 broad categories: Non-small cell lung cancer (NSCLC) and small cell lung cancer (SCLC). NSCLCs account for around 85% of cases and are grouped together as they behave in a similar manner.

The three main types of NSCLC are:

- Adenocarcinoma – 40% of NSCLC, derived from mucous-producing cells
- Squamous cell cancer – 15% of NSCLC, often occurring in the main airways
- Large cell carcinoma – 25% of NSCLC, more aggressive than the other forms and can be found anywhere in the lungs.

Rarer forms of NSCLC include lung carcinoid tumours, sarcomas and lymphomas.

SCLC comprises approximately 20% of lung malignancies, are neuroendocrine in nature and tend to have metastasised by the time of presentation. Secretory sequelae include Lambert-Eaton myasthenic syndrome, syndrome of inappropriate antidiuretic hormone (SIADH) or Cushing's syndrome.

Pleural malignancy, in the form of mesothelioma is associated with asbestos exposure and prevention focusses on reducing this exposure.

Secondary lung malignancies most commonly originate from the breast, colon, rectum, head and neck, kidneys and reproductive organs.

What are the pulmonary sequelae of lung malignancies?

Pulmonary sequelae are due to local or systemic effects of the tumour. Locally, growth of lung cancer may narrow airways, causing distal hyperinflation and wheeze. Ultimately, complete airways obstruction can occur, causing V/Q mismatch, hypoxia and regional lung collapse or consolidation. Tumour can invade into vasculature leading to acute pulmonary haemorrhage. Further, increased mucous production secondary to lung cancer can lead to airways obstruction or a clinical picture resembling bronchiectasis.

Malignancy in the pleural cavity can increase pleural fluid production or reduce drainage, causing an exudative pleural effusion.

Hormone secretion from lung malignancies can have pulmonary effects. For example, lung carcinoid can lead to carcinoid syndrome which may include bronchoconstriction leading to an obstructive airways picture.

What are the systemic effects of lung malignancy?

Generalised systemic complications include anorexia, weight loss and functional decline. This is likely due to a combination of central anorexia and catabolic effects of pro-inflammatory cytokines such as TNF, IL-1 and IL-6. Lung malignancy also creates a prothrombotic state, increasing the risk of venous thromboembolism.

Squamous cell carcinoma is associated with secretion of parathyroid hormone-related protein, leading to hypercalcaemia. Similarly other lung malignancy can lead to hypercalcaemia due to bony metastasis.

SCLC can lead to a variety of neuroendocrine sequelae. Ectopic ACTH or ADH secretion can result in Cushing's syndrome or SIADH respectively. SCLC can also lead to autoantibodies against voltage-gated calcium channels, leading to Lambert-Eaton myasthenic syndrome.

Metastasis from lung primary most commonly spread to the brain, liver and bone leading to features of bony pain, jaundice and neurological sequelae such as headache, dizziness and seizures. Local metastasis can also lead to compression of the superior vena cava leading to headache, facial swelling and plethora and shortness of breath.

Tell me about Lambert-Eaton myasthenic syndrome.

This is a paraneoplastic condition, most associated with SCLC. It arises due to autoantibodies against voltage-gated calcium channels (VGCC) in the neuromuscular junction, autonomic nervous system and cerebellum. Within the neuromuscular junction, calcium is required for synaptic vesicle fusion and release of acetylcholine, the VGCC autoantibodies reduce its availability thus reducing skeletal muscle contraction.

Features are of a symmetrical proximal weakness, affecting legs more than arms. Unlike myasthenia gravis, facial and eye muscle involvement is rare. Approximately 75% of patients will have concomitant autonomic features with constipation, blurred vision, orthostatic hypotension and dry mouth.

Diagnosis is made based on the presence of VGCC antibodies (present in 85%), nerve conduction studies and electromyography. Nerve conduction studies demonstrate normal conduction velocities and latency with small amplitude. Unlike myasthenia gravis, repeated stimulation increases acetylcholine release and therefore amplitudes increase on repetition.

Definitive treatment involves diagnosis and management of the underlying tumour; however, symptomatic relief may be achieved using a combination of immunosuppression, immunoglobulins and plasma exchange.

Which patients are suitable for tumour resection?

The two main considerations here are those of operability and those related to the patient. Lung cancer is considered operable if it is T1–3 N0–1 M0 NSCLC. T4 and N2 NSCLC may be operable in conjunction with chemo-radiotherapy or biological therapy. Limited stage SCLC may be considered for surgery. Lobectomy is preferred, however, pneumonectomy may be required for more central tumours.

Clear guidelines have been published outlining the strategy for patient assessment for surgery. These guidelines consider age, respiratory function, cardiovascular function, performance status and nutrition.

With respect to respiratory function, a patient is considered low risk for lobectomy if they have a post-bronchodilator $FEV_1 > 1.5L$ and low risk for pneumonectomy if their FEV_1 is $> 2L$. This group of patients require no further pulmonary testing.

Those who have an FEV_1 below this will require further testing in the form of pulmonary function tests, shuttle-walk tests or cardiopulmonary exercise testing. Recent NICE guidance considers patients high-risk if they have a $VO_2 \text{ max} < 15 \text{ ml/kg/min}$, a shuttle walk test $< 400 \text{ m}$ or a postoperative predicted FEV_1 or $TLCO < 30\%$. These may be considered for more limited resection.

It is important to note that being high-risk is not a contraindication for radical surgery; rather, patients should be fully informed of the associated morbidity and mortality. NICE guidelines recommend the use of thoracoscopes to assess patient risk of mortality; however, evidence on its predictive accuracy is variable.

How are postoperative FEV_1 and $TLCO$ estimated?

Predicted postoperative FEV_1 ($ppoFEV_1$) can be estimated through lung segment counting. There are 9 segments on the left and 10 on the right. The following formula can be applied:

$$ppoFEV_1 = preoperativeFEV_1 \times \frac{(19 - \text{segments to be resected})}{19}$$

Perfusion scintigraphy can give a more accurate measure of $ppoFEV_1$ by indicating perfusion to each lung segment. By multiplying preoperative FEV_1 by the percentage of whole lung perfusion contributed by the non-operated lung, an estimate of $ppoFEV_1$ is gained. This method is particularly useful when it shows minimal loss of FEV_1 due to tumour compression of the vascular supply to the lung to be resected.

$TLCO$ is calculated using scintigraphy in a similar manner to FEV_1 shown above. FEV_1 and $TLCO$ measure different characteristics of respiratory function, therefore, an acceptable result in one does not equate to an acceptable result in the other.

How would you anaesthetise a patient undergoing pneumonectomy for lung malignancy?

I would split my management into preoperative, intraoperative and postoperative management.

Preoperatively I would take a full anaesthetic history, focussing specifically on the cardiorespiratory systems. Patients undergoing this surgery should have been seen in preoperative assessment clinic; therefore, I would review the clinic notes and the results of investigations. I would review the most recent bloods, ensure that there were 2 units of blood cross-matched and that a high dependency bed was available. I would ensure that the patient was fully consented for the anaesthetic and that there were no questions from the preoperative clinic visits that were outstanding.

In the anaesthetic room I would complete standard preoperative checklists and insert wide-bore intravenous access. I would insert a thoracic epidural in the patient awake and then induce the patient with a balanced anaesthetic. Following rigid bronchoscopy, I would insert a double lumen tube (DLT) except in the case of difficult airway, when I would use a bronchial blocker. For left pneumonectomy I would insert a right DLT, paying careful attention to positioning of the Murphy's eye. Following intubation I would insert an arterial line, central line, urinary catheter and instigate core temperature monitoring. My choice of antibiotic would be based on hospital guidelines.

Maintenance of anaesthesia would be achieved either through volatiles or a TIVA technique. I would instigate lung protective ventilation with sufficient PEEP. I would commence the epidural infusion intraoperatively and continue it into the postoperative period. Haemodynamically, I would use vasopressors to offset hypotension secondary to epidural infusion and would employ a restrictive fluid strategy to minimise the risk of postoperative pulmonary overload. I would be particularly vigilant during the time of pulmonary artery clamping as this may lead to right ventricular failure and cardiovascular collapse. The patient will be placed in the lateral decubitus position, therefore, I would pay careful attention to their neck position, pressure points and eyes. Normothermia would be maintained with forced air and fluid warming and venous thromboembolism risk minimised with intermittent pneumatic compression devices.

Postoperatively I would extubate the patient normothermic, fully awake and with sufficient analgesia on board. They should be managed postoperatively in a high dependency area.

Interstitial Lung Disease

How would you classify interstitial lung disease (ILD)?

Classification of ILD has recently been standardised internationally by the American Thoracic Society/ European Respiratory Society (ATS/ERS) multidisciplinary panel. ILD is now broadly grouped into 4 categories:

- ILD of known cause/ association – drug related, connective tissue disorders or occupational
- Idiopathic interstitial pneumonias – subclassified into major, rare and unclassifiable
- Granulomatous disease
- Other forms of ILD.

Can you give some examples of causes of ILD?

A mnemonic here can help you remember this list of conditions

Common causes of ILD can be split into those that are upper lobe predominant and those that are lower lobe predominant (Table 1.8.4):

Table 1.8.4 Common causes of ILD

Upper lobe predominant	Lower lobe predominant
C Coal workers pneumoconiosis	C Cryptogenic fibrosing alveolitis (now idiopathic pulmonary fibrosis)
H Histiocytosis	A Asbestosis
A Ankylosis spondylitis/ allergic bronchopulmonary aspergillosis	R Rheumatoid arthritis
R Radiation	D Drugs (bleomycin/ methotrexate/ amiodarone)
T Tuberculosis	S Systemic sclerosis
S Silicosis/ sarcoidosis	

What results would you expect from pulmonary function testing in a patient with interstitial lung disease?

Patients with interstitial lung disease will have a restrictive defect on pulmonary function testing. Forced vital capacity (FVC) and Forced expiratory volume in one second (FEV₁) may be normal in the early stages of disease but will both be reduced below 80% of predicted in established disease. FEV₁/FVC ratio will be maintained within the normal range.

Diffusing capacity of carbon monoxide (DLCO) and the carbon monoxide transfer coefficient (KCO) will also be reduced, indicating that fibrosis has increased the width of the diffusion barrier into the pulmonary vascular system.

What is the difference between DLCO and KCO and why is knowledge of both results important?

This is a dry question, but is designed to determine whether the candidate truly understands the tests that they are requesting and interpreting.

Generally speaking, DLCO is a measure of the global lung conductance of inspired gas to red cells. Although not strictly correct, DLCO is equivalent to the following equation, where VA is alveolar ventilation:

$DLCO = KCO \times VA$

KCO, therefore, is an index of carbon monoxide diffusion efficiency and indicates an abnormal alveolocapillary membrane.

Reductions in DLCO can therefore occur due to

- Reduction in diffusion efficiency – interstitial lung disease, emphysema
- Reduction in alveolar ventilation – airways obstruction, bullae, pneumonectomy, hypoventilation.

What are the key considerations when anaesthetising a patient with interstitial lung disease and how should they be anaesthetised?

Patients with ILD should receive a full anaesthetic assessment with a focus on the cardiorespiratory systems. Many of these patients are chronically hypoxic and a knowledge of their normal oxygen saturations will provide a target under anaesthesia.

Postoperative pulmonary complications represent the primary risk for patients with ILD and should be a primary focus of the preoperative assessment. A scoring system such as the Assess Respiratory Risk in Surgical Patients in Catalonia (ARISCAT) score can help in this regard. Further information on respiratory risk can be gained through baseline arterial gas analysis and pulmonary function tests.

Comorbid conditions should be identified and optimised. In particular, meticulous cardiovascular assessment should also be performed. Many patients with significant ILD may have pulmonary hypertension and right heart failure. These patients are at high risk of decompensation under anaesthesia and would be best managed with a regional technique if possible. Patients with significant ILD should all have a preoperative transthoracic echocardiogram and referral to cardiology if there is evidence of significant pulmonary hypertension. Cardiopulmonary exercise testing should also be considered if available, as this will aid risk assessment and counselling of the patient for anaesthesia.

When conducting anaesthesia, if possible, a neuraxial or regional technique should be favoured as this will least impact the patient's respiratory function. In those for whom general anaesthesia is unavoidable, an arterial line should be inserted before induction. Ventilator settings should use an FiO_2 that achieves normoxia for the patient.

Lung protective ventilation is recommended in this population as it most closely matches normal ventilatory mechanics in patients with ILD and minimises increases in right ventricular afterload. Peak inspiratory pressures and PEEP should be minimised whilst maintaining an awareness that high CO_2 and low O_2 will increase pulmonary vascular resistance and increase strain on a likely already struggling right ventricle. Patients may benefit from cardiac output monitoring to optimise preload. Normothermia, adequate analgesia and a minimisation of vasopressors will reduce increases in pulmonary vascular resistance.

It should be considered that this patient population may be on long-term steroids and the dose should be increased for the perioperative period according to sick-day rules. Postoperative management for these patients should be in a high dependency or critical care setting; however, reasonable postoperative expectations and appropriate ceilings of care should have been discussed with the patient and implemented before the patient undergoes surgery.

Further Reading

BTS guidelines: Guidelines on the selection of patients with lung cancer for surgery. *Thorax*. 2001; 56(2): 89–108.

Kevin LG, Barnard M. Right ventricular failure. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2007; 7 (3): 89–94.

Lung cancer: Diagnosis and management
[Nice guideline NG122]. 2024. www.nice.org.uk

Travis WD, Costabel U, Hansell DM, King TE, Jr., Lynch DA, Nicholson AG, et al. An official American Thoracic Society/ European Respiratory Society statement:

Update of the international multidisciplinary classification of the idiopathic interstitial pneumonias. *American Journal of Respiratory and Critical Care Medicine*. 2013; 188(6): 733–748.

1.9.1 Renal Failure – Rosada Jackson and Jessie R Welbourne

Acute renal failure is covered in Section 3.2.6. 'Renal Replacement Therapies'.

A 40-year-old woman presents for formation of an arterio-venous fistula to allow dialysis. She has established end-stage renal failure secondary to diabetes.

Describe your approach to the management of this patient.

A structured approach is essential. Start off with an opening statement about the condition and then classify.

Patients with renal failure have a spectrum of problems affecting all of their organ systems.

Their renal failure will also affect their drug handling.

A full preoperative assessment must be made, with particular attention to comorbidities and biochemical state. The preoperative assessment and knowledge of the effects of renal failure may alter induction, maintenance of anaesthesia and postoperative care.

Can you expand on the effects of renal failure?

Yes, considering each system in turn:

- Cardiovascular: ischaemic heart disease, hypertension, congestive cardiac failure, pericarditis and endocarditis are associated with renal failure
- Respiratory: pulmonary oedema due to fluid overload and pneumonia due to impaired immunity or immunosuppressive medication
- Haematological: coagulopathy, specifically platelet dysfunction and prolonged bleeding time, anaemia due to chronic disease, iron deficiency, reduced erythropoietin production and shortened red cell lifespan due to uraemia
- Endocrine: diabetes and parathyroid dysfunction which causes hypocalcaemia and hyperkalaemia
- Gastrointestinal: peptic ulceration and gastric reflux due to delayed emptying
- Immune: impaired immunity with a propensity for infection
- Nervous: neuropathy (motor, sensory and autonomic) and myopathy.

What are the main causes of chronic renal failure?

Chronic renal failure can be caused by systemic diseases which affect the kidney. Diabetes and hypertension account for approximately two-thirds of cases. Other

systemic diseases that cause CKD include autoimmune vasculitides, drugs, amyloidosis, and polycystic kidney disease.

Diseases may also specifically target the kidney. For example, glomerular disease such as IgA nephropathy or focal segmental glomerulosclerosis (FSGS), tubulointerstitial disease e.g. obstruction or urinary-tract infections, or vascular disease e.g. fibromuscular dysplasia.

Approximately 15% of cases of ESRF are idiopathic.

How is chronic renal failure classified?

The 2021 NICE guidelines classify chronic kidney disease using the CGA staging which takes into account both the underlying pathology and the decline in renal function.

C refers to the cause of renal failure, and is classified by the presence or absence of systemic disease as well as the location of the pathology within the kidney. The most common identified cause is diabetes mellitus (19.8%) followed by glomerulonephritis (10.3%).

G refers to the eGFR category (G1 eGFR >90 ml/min, G2 eGFR 60–89 ml/min, G3a eGFR 45–59 ml/min, G3b eGFR 30–44 ml/min, G4 eGFR 15–29 ml/min, G5 'kidney failure' eGFR <15 ml/min).

A is the assigned albuminuria category (A1 albumin:creatinine ratio (ACR) < 30, A2 ACR 30 – 300, A3 ACR >300).

What are the modes of renal replacement therapy?

The number of people with ESRF in the UK is steadily increasing. The number of patients receiving RRT for ESRF at the end of 2019 was 68,111. Over half of these had a functioning transplant (56.8%), followed by in-centre haemodialysis (35.8%), peritoneal dialysis (5.4%) and home haemodialysis (2%).

What details in the preoperative history are important?

The preoperative assessment of patients with chronic renal failure should cover the aetiology of their renal failure, their current and previous modes of renal replacement therapy, a history of their lines and fistulas (including why they failed), their baseline creatinine, dry weight and their normal blood pressure.

Cardiovascular risk assessment is essential due to increased incidence of hypertension, MI, CCF, CVA (both ischaemic and haemorrhagic), peripheral vascular disease and arrhythmias. A thorough review of their current medication will identify drugs such as anticoagulants, anti-platelets, DMARDS, anti-hypertensives, steroids and diabetic agents, all of which will have a bearing on their anaesthetic management.

There is a high incidence of both Type 1 and Type 2 diabetes so it is important to establish their insulin regime and usual blood glucose range. Some patients will have had a kidney-pancreas-transplant, so although their blood glucose may now be controlled, they will have end organ damage and increased cardiovascular risk.

The timing of preoperative dialysis will particularly affect potassium and fluid status. Many centres favour 24 hours post-dialysis as the optimal time for surgery. A patient immediately post-dialysis may be fluid depleted, worsened by fasting for surgery, whereas fluid overload is more likely if dialysis is overdue.

What examination findings are of particular importance?

Assessment of fluid status is important, by looking at heart rate, blood pressure, CVP or JVP, extent of oedema and weight trends. Fluid status should be assessed as fluid overload, pulmonary oedema, and pleural effusions may be present and should be treated preoperatively.

What investigations are important?

Here is a golden opportunity to show you are safe, which is an important fact to convey in any anaesthetic exam!

Of particular relevance in renal failure is the most recent potassium level. If there has been absent or inadequate dialysis and poor renal function, this is likely to be raised. This may result in significant arrhythmias perioperatively. If the serum potassium is >6.0 mmol/L then surgery should be delayed as correcting the hyperkalaemia is the priority.

A full blood count may reveal a normochromic, normocytic anaemia. In chronic renal failure this is secondary to decreased erythropoietin secretion.

The 2,3-DPG concentration in the red cell is increased in chronic anaemia, so aiding the offloading of oxygen to the tissues. It is for this reason that some anaesthetists will proceed with surgery when the patient's haemoglobin concentration is as low as 6 g/L. However, the predicted operative blood loss, which may be affected by underlying coagulopathy and platelet dysfunction, will often dictate the need for a higher preoperative haemoglobin concentration.

Platelet count and function may be impaired and there may be a coagulopathy secondary to uraemia. Significant disturbances should be corrected preoperatively. It is important to note that the tests of coagulation and platelets may be normal, but it is often the bleeding time that is prolonged.

The severity of uraemia is important, and this has multi-system effects such as:

- Pericarditis
- Encephalopathy
- Peripheral neuropathy
- GI effects: anorexia, nausea, delayed gastric emptying, vomiting and diarrhoea
- Dermatological: dry skin, bruising, pruritis
- Restless leg syndrome
- Fatigue
- Coagulopathy secondary to platelet dysfunction
- Erectile dysfunction, amenorrhoea.

Magnesium levels may be high or low depending on the adequacy of filtration and this may predispose to cardiac arrhythmias.

Valvular dysfunction, pericardial effusions and atheromatous disease, causing ventricular dysfunction are common in patients with renal failure. A recent 12-lead ECG must be reviewed. Often patients will have had a series of echocardiography investigations over time, allowing the clinician to see a trend in the patient's cardiac function.

What factors will you consider when anaesthetising this patient?

At this point, try to visualise this patient in your anaesthetic room and what your normal options include.

Vascular access surgery for renal dialysis may be done under a regional or general anaesthetic, and the choice depends on the surgical site, patient risk factors (including coagulation status) and the patient's preference. A regional technique avoids exposure to anaesthetic agents and the associated risks of a general anaesthetic, as well causing localised vasodilatation which aids surgery and improves postoperative graft patency.

If proceeding with a general anaesthetic, induction doses should be given judiciously while palpating the pulse due to the potential for significant hypotension. The threshold for endo-tracheal intubation should be lower in these patients due to autonomic dysfunction and the increased incidence of reflux.

Care must be taken to position cannulae away from potential fistula sites and to avoid arterial lines if possible, to preserve future dialysis access. Existing dialysis lines should not be accessed unless in an emergency, and they must be aspirated first to remove the heparin used to 'lock' the line e.g. protect it from obstruction with thrombus. BP cuffs also must not be positioned on the same limb as a fistula since they disrupt blood flow through the fistula increasing the chance of thrombosis. Fluid balance is key, therefore in oligo-anuric patients routine IV fluids should be limited to 500 ml normal saline. In diabetic patients, hourly BMs are required to maintain normoglycaemia which aids wound healing and reduces the risk of infection.

Finally, renal patients may be frail with significant muscle wasting and limited mobility therefore positioning for surgery must be done attentively to avoid causing skin tears and pressure sores.

What factors affect your choice of drugs?

Drugs that are renally excreted will have a prolonged duration of action, for example morphine. Morphine is metabolised in the liver and excreted in urine or bile. The metabolites morphine glucuronide and morphine sulphate are renally excreted. Although small single doses of morphine are safe, fentanyl is favoured for regular analgesia.

Many muscle relaxants are renally excreted so the duration of action for these drugs is increased. The duration of action of atracurium is not affected by renal failure as it is metabolised by Hoffman degradation in the plasma and so it is a common choice when muscle relaxation is required.

Chronic metabolic acidosis in renal failure affects drug ionisation. Low albumin and decreased levels of glycoproteins also reduces binding capacity. This increases the free fraction of available drug, and also prolongs the duration of drug action particularly for highly protein-bound drugs such as benzodiazepines.

NSAIDs should be avoided as they inhibit prostaglandin mediated intrinsic renal vasodilatation and are associated with GI bleeding. Suxamethonium should also be avoided as its use will result in a rise in serum potassium, which could be dangerous in renal patients who already tend to be hyperkalaemic. A single dose of suxamethonium may increase the potassium by 0.5 to 1 mmol/L.

Anaesthetic agents will cause decreased SVR and some depression of myocardial function. This results in reduced blood pressure, reduced renal blood flow and glomerular filtration rate. It is therefore important to administer intravenous anaesthetic agents cautiously to minimise these effects. Some inhalational agents with fluoride groups have been experimentally shown to be nephrotoxic, but the significance of this in practice is not thought to be significant.

Remifentanyl undergoes ester-hydrolysis in the plasma and has a fixed context sensitive half-life. Its dosing and duration of action are therefore unaffected by

renal dysfunction. It can provide predictable control over the onset and offset of anaesthesia.

Overall, a balanced anaesthetic should be given, using a combination of short-acting and non-cumulative opioids, simple analgesia, local anaesthetics and cautious use of intravenous and inhaled agents.

What complications may occur perioperatively?

All dialysis patients are at an increased risk for cardiovascular complications, particularly arrhythmias and hypotension. Particular care should be taken with anaesthetic agents as hypotension may result. The combination of autonomic dysfunction and arteromatous disease impairs endovascular responsiveness, which means that renal failure patients are at high risk of vasodilatory hypotension. Hypotension should be treated with low volume fluid challenges (100 ml). Alpha adrenoreceptor agonists can produce vasoconstriction of the renal vasculature and so should be avoided in patients with residual renal function. Ephedrine is better tolerated in renal failure but should be used with caution as hypertensive surges and tachyarrhythmias may also occur.

Hypoxia should be rapidly corrected with increased FiO₂ and use of PEEP in the first instance. Other causes including reduced oxygen carriage due to anaemia should be assessed and corrected if needed.

What care will the patient need postoperatively?

The normal safety measures for postoperative care should be followed and a level 2 care environment should be considered. Monitoring should include pulse oximetry, non-invasive blood pressure, oxygen saturations, respiratory rate and estimation of fluid status. Bedside estimation of haemoglobin may be performed and there should be a low threshold for its use in recovery in view of the coagulopathy associated with renal failure and resultant blood loss. Postoperatively, serum electrolytes and routine biochemistry should be checked with particular attention to the serum potassium because if this is raised urgent renal replacement therapy may be indicated. Analgesia should be provided with a balance of agents, including local anaesthetic, paracetamol and opioids. Caution is needed with opioids to ensure the dosing interval should be increased and the respiratory rate monitored. Increasingly, vascular access surgery for renal dialysis is being done as a day-case procedure provided the patient meets criteria, so this should be considered as an option.

Further Reading

Bradley T, Teare T, and Milner Q. Anaesthetic management of patients requiring vascular access surgery for renal dialysis. *British Journal of Anaesthesia. Education*. 2017; 17 (8): 269–274.

Chronic kidney disease: Assessment and management, *NICE guideline (NG203)* August 2021

Craig RG, Hunter JM. Recent developments in the perioperative management of adult patients with chronic kidney disease. *British Journal of Anaesthesia*. 2008; 101 (3): 296–310

Milner Q. Pathophysiology of chronic renal failure. *British Journal of Anaesthesia. CEPD Reviews*. 2003; 3(5): 130–133.

National Kidney Foundation. The National Kidney Foundation Kidney Disease Outcomes Quality Initiative (KDOQI). www.kidney.org/professionals/kdoqi/

UK Renal Registry (2021) UK Renal Registry 23rd Annual Report – data to 31/12/2019, Bristol, UK. Available from renal.org/audit-research/annual-report.

Cardiothoracic

2.1.1 Anaesthesia for Cardiac Surgery, Cardiopulmonary Bypass and Cardioplegia – Jessie R Welbourne and Katharine Smurthwaite

Don't panic if this topic comes up. Your examiners will either not be cardiac anaesthetists or are not allowed to use specialist knowledge in the exam. This is a large subject, and you only need to demonstrate that you have a working knowledge of the normal principles and practice of cardiac anaesthesia.

A 68-year-old man presents for elective aortic valve replacement with coronary artery bypass grafting.

Cardiac anaesthesia could be linked with many topics. Physiology of the valvular defect is highly likely. This topic is covered in Section 1.2.1. 'Preoperative Assessment and Management of Patients with Cardiac Disease' and Section 1.2.6. 'Valvular Defects'.

What areas in the history are of particular relevance to your anaesthetic management plan?

This could be an entire viva topic in itself. Keep it simple and structured, and the examiners will direct you to the area they wish to discuss.

Cardiac anaesthesia is inherently high risk. Mortality can vary from approximately 2–10% depending on the operation. A full anaesthetic assessment is required to allow risk stratification (commonly EUROSCORE II), planning of care and appropriate discussions with the patient.

Areas which require a thorough review include:

- History:
 - Current cardiac disease, especially ischaemia and cardiovascular stability. These indicate the urgency of surgery. CCF, arrhythmias and atherosclerosis – which is associated with an increased stroke risk
 - Modifiable risk factors and comorbidities including smoking, diet, exercise, nutritional status, diabetes
 - Drugs, especially antiplatelets, ACE inhibitors, β -blockers, statins, anticoagulation.
- Investigations:
 - Echo: preoperative cardiac function
 - ECG: a baseline ECG is paramount

- Any recent cardiac catheterisation with occluded vessels identified and treatment provided (medication or stenting)
- Bloods including
 - Renal function: RRT is required in approximately 5% (1 in 20) patients and is associated with up to 50% mortality. Those with CKD are more likely to develop a post-op AKI.
 - FBC: anaemia is independently associated with adverse outcomes.
- Carotid Doppler ultrasound scan to help assess stroke risk.

Would you offer a 'pre-med' to a patient such as this?

Preoperative sedatives can be useful to reduce the cardiovascular effects of anxiety, including the effects of increased catecholamines. They can, however, be associated with increased postoperative delirium and confusion and therefore may be avoided in patients on enhanced recovery pathways requiring early extubation. Currently in our unit, pre-meds are decided on an individual patient basis.

Describe how you would induce anaesthesia in this patient.

It pays to visualise what you do in practice and talk it through step by step. An examiner will not expect you to have had extensive first-hand experience of cardiac anaesthesia, but knowledge of the principles of managing patients with cardiac disease will be required.

The primary aims of anaesthesia induction are to maintain cardiovascular stability. The pressor response to intubation should be obtunded; however, myocardial suppression and sudden decreases in systemic vascular resistance should be avoided.

Prior to induction, all anaesthetic drugs, including inotropes, vasopressors and vasodilators, and equipment should be ready. Staff briefings and checklists should be complete and full AAGBI monitoring in situ, with at least one large bore intravenous access. Invasive arterial monitoring should be secured, and the patient fully preoxygenated.

Induction is commonly achieved with high-dose opioid such as 10–15 mcg/kg of fentanyl and carefully titrated propofol. Alternatively, total intravenous anaesthesia (TIVA) with propofol and remifentanyl may be chosen. Any adverse haemodynamic changes should be treated promptly. Following induction, a muscle relaxant is given, allowing intubation.

Following intubation, a central venous catheter is inserted to provide venous pressure monitoring and access for inotropic agents. A pulmonary artery catheter sheath may also be considered. A urinary catheter and central temperature probe should be inserted. Processed or raw EEG monitoring may be appropriate, as awareness during cardiac surgery is a recognised risk. A transoesophageal echo probe may also be required (usually during valve operations). Antibiotics and tranexamic acid should be administered.

Which volatile agent would you use?

Common agents used are sevoflurane or isoflurane. While theoretically isoflurane has been implicated in coronary steal, where blood flow is directed away from ischaemic

areas due to vasodilatation of healthy coronary arteries, it is not thought to be clinically relevant. Desflurane may be best avoided as it can cause sympathetic stimulation and tachycardia.

What can you tell me about ischaemic preconditioning?

Don't panic if you don't know much on the subject, if you get asked this you are doing well!

Ischaemic preconditioning involves the induction of short ischaemic periods prior to a period of prolonged tissue ischaemia. The brief ischaemic periods have been shown to render the tissue more resistant to the adverse effects of prolonged ischaemia or reperfusion. The mechanism underlying this phenomenon is thought to involve reactive oxygen species triggering the release of endogenous agents such as adenosine and nitric oxide.

Studies have shown that, in addition to brief ischaemic periods, pharmacological agents, particularly the volatile agents can be used perioperatively to precondition the myocardium via similar pathways.

What is cardiopulmonary bypass, and what are its functions?

Cardiopulmonary bypass is an extracorporeal circulation that takes over the role of the heart and lungs, allowing surgery to proceed while allowing continued oxygenation and circulation of the blood.

A critical difference to normal flow is that cardiopulmonary bypass flow is non-pulsatile. Other functions include allowing surgical access to a bloodless and motionless heart while providing the patient with oxygenated blood at a predetermined temperature and pressure. Carbon dioxide can also be removed by the bypass machine. Anaesthetic vapour or TIVA, and vasopressors or vasodilators may be added. The cardiopulmonary bypass machine can also enable rapid cooling or heating of blood whenever required, controlling temperatures between 15 and 37°C.

What are the key steps in instituting cardiopulmonary bypass?

The skin is prepared and draped by the surgical team, and a median sternotomy is performed soon after skin incision. The surgical stimulation of this and perhaps concurrent harvesting of leg veins may result in a surge in blood pressure and pulse rate, which will need intervention without producing negative inotropy. A short-acting opioid is often used.

Prior to commencing bypass, a baseline arterial blood gas is performed, checking basic haematological and biochemical parameters. A baseline activated clotting time (ACT) is performed, and if the patient has been on an antiplatelet, platelet mapping may be performed. Before the arterial and venous bypass cannulae are inserted, the patient will need to be fully anticoagulated. Heparin should be administered centrally at a dose of 300 IU/kg. An activated clotting time should be checked after 3 minutes to ensure adequate anticoagulation. The ACT should be above 400 or 4 times normal, which is 100–140 seconds, to allow bypass to safely proceed.

The extracorporeal circuit will have been primed, usually with a crystalloid, heparin, or occasionally mannitol, resulting in a fall in haematocrit during bypass. Blood may also be considered if the haematocrit is low; however, it is mainly used in paediatric cardiac

surgery. The systolic pressure should be 80–100 mmHg to reduce the risk of aortic dissection at the time of aortic cross-clamping. Vasodilators may be required to achieve this and should be on hand. The aorta is usually cannulated first to allow volume resuscitation if required, followed by the right atrium. Bypass is then initiated.

Cardioplegia solution may be used. Once the patient is on bypass, the ventilator can be turned off and anaesthesia maintained by either TIVA or a volatile agent via the bypass circuit.

What are the key components of the cardiopulmonary bypass circuit?

A basic understanding is all that is required. Remember to keep it simple – it's just a circuit with tubes in and out, a pump, an oxygenator and a heat exchanger. Learn to draw a simple diagram of this.

Venous cannulae from the superior or inferior vena cava, or right atrium drain blood into a reservoir. Filtered blood from the suction can be added to the reservoir. The blood is passed through an oxygenator, normally a membrane oxygenator, and a heat exchanger, before being pumped through a filter into the arterial cannula, which is normally inserted into the ascending aorta distal to the cross-clamp, returning blood to the patient's circulation.

What do you know about blood pumps?

The pumps used are either roller or centrifugal pumps and are required to deliver flow against resistance. They must avoid any areas of blood stasis potentially causing emboli or turbulence, which may cause haemolysis. The pumps deliver a flow of 2.4 l/min/m^2 , to correspond with a normal cardiac index. In contrast to the physiological circulation, the flow delivered is non-pulsatile.

You mentioned membrane oxygenators, do you know any other types of oxygenators and their advantages and disadvantages?

Yes, I am aware of membrane and bubble oxygenators. Membrane oxygenators are most commonly used. They contain hollow fibres giving a large surface area for gas exchange, which occurs down concentration gradients. Bubble oxygenators are when the gases are bubbled through the blood, but this leads to increased risk of air embolism and is less commonly used.

What are the complications associated with cardiopulmonary bypass?

The complications of cardiopulmonary bypass (CPB) relate to the circuit and to perfusion.

Circuit-related complications include:

- Obstruction of the cannulae
- Failure of the oxygenator
- Inadequate anticoagulation causing emboli
- Aortic dissection
- Air embolism
- Haemorrhage – intra- or post-op

Complications associated with perfusion include:

- Hypothermia
- Fluid overload
- Myocardial stunning
- Coagulopathy: occurs particularly with pump time greater than 2 hours
- SIRS: prolonged bypass can lead to release of cytokines
- Electrolyte and acid–base disturbances
- Cerebrovascular events: 1–5% of patients affected, ranges from a transient postoperative cognitive dysfunction to a major disabling stroke.

What do you understand by the term myocardial preservation?

Myocardial preservation or protection aims to preserve myocardial function and prevent cell death. Cardioplegic solutions are used to achieve this. They are administered to the myocardium to cause a diastolic electromechanical arrest. A cold solution can be used to reduce metabolic rate. Cardioplegia solution is stored at 4 °C then injected rapidly under pressure into the aortic root (antegrade) or coronary sinus (retrograde), making sure no air bubbles enter the coronary circulation. Cold cardioplegia put into the pericardium and chambers achieves a uniformly cooled heart. Further doses of cardioplegia are required every 20 minutes or when electrical activity returns. The core body temperature is actively cooled to a target value of between 28 °C and 32 °C. Modifications of this technique are used in different centres.

What is the composition of cardioplegia solution?

There are a number of different formulations. These are crystalloid or blood-based solutions to which ingredients are added. Ringer's lactate, dextrose and saline/dextrose solutions have all been used. Potassium chloride is the agent used to induce cardiac arrest. It is added at a concentration of 20 mmol/L to depolarise myocardial cells, with procaine as a membrane-stabilising agent. Magnesium may also be added.

What is the sequence of events when coming off bypass?

There must be clear communication between the surgeon, anaesthetist and perfusionist. The patient's temperature should be returned to 37 °C. This is achieved via CPB and the heat exchanger, in conjunction with non-invasive methods, such as forced-air warmers, warming mattresses and warmed intravenous fluid. This may lead to hypotension or metabolic abnormalities with cardiovascular instability.

The potassium should be 4 to 5 mmol/L and the haematocrit should be greater than 24%. Acid–base should ideally be normalised. A sinus rate of 70 to 100 beats/min is desirable. Rewarming may cause malignant arrhythmias requiring DC cardioversion. Epicardial pacing wires will almost always be required for valve procedures and may also be inserted if there are difficulties achieving a regular rhythm. De-airing of the heart should also occur. The venous pump is gradually restricted, allowing venous return back into the right atrium. As cardiac activity returns, blood begins to circulate into the pulmonary vasculature, and mechanical ventilation is recommenced. If used, the inhaled volatile agent is also recommenced. Once the ventricles are contracting well, the remaining circulating volume is returned to the heart.

Protamine 1 mg per 100 units of heparin is administered by slow infusion to reverse the effects of heparin, but only when surgically indicated, and at this point the perfusionist must turn off the suction. Protamine may precipitate systemic hypotension, pulmonary vasoconstriction and anaphylaxis so must be given slowly and cautiously.

What investigations should be performed post-bypass?

Intraoperatively a repeat arterial blood gas is taken giving an overview of biochemical and haematological parameters. Viscoelastic coagulation testing is useful to direct blood product requirements. Once on ICU, serum samples should be taken for a full haematological, biochemical and coagulation screen.

What are the important features of postoperative care?

The patient should be looked after in a critical care environment with invasive monitoring and appropriate experienced nursing and medical care.

The patient will usually require a period of sedation and ventilation postoperatively. The length of time will depend on which anaesthetic and analgesic drugs are used and whether the patient is stable. 'Fast-track' cardiac anaesthesia, meaning extubation within 6 hours after cardiac surgery, has been established as routine in many cardiac centres.

The patient should be normothermic, haemodynamically stable and have any acid-base or electrolyte disturbances corrected prior to extubation. Most patients will require volume expansion as they 'warm up', and a high urine output as a result of the filtration of the 'pump prime' can lead to hypokalaemia. Bleeding from the chest or pericardial drains in the postoperative period will need investigating, necessitating the administration of blood, platelets, FFP or more protamine, directed by point-of-care testing. Cardiac tamponade should be considered and surgical exploration may be required.

Once extubated, analgesia can be maintained using patient-controlled opioid analgesia and regular paracetamol.

What is 'minimally invasive cardiac surgery'?

Minimally invasive cardiac surgery (MICS) is an umbrella term for a collection of surgical techniques which avoid the conventional midline sternotomy approach. It can be used for a variety of procedures including one or two vessel coronary bypass grafting, selected valve procedures and atrial fibrillation ablation surgery. Approaches can include mini-sternotomy, mini-thoracotomy and video-assisted thorascopic surgery (VATS). Patient selection is key, and one-lung ventilation may be required. Cardiopulmonary bypass may be utilised using a peripheral approach, or alternatively, an 'off-pump' approach may be appropriate.

Off-pump surgery potentially reduces both ICU and hospital stay through reduced blood loss and reduced postoperative analgesic requirements, allowing frailer patients earlier return to normal activities. It does, however, also have associated risks. It is more technically complicated and only carried out in specialised centres. Peripheral cannulation for bypass has associated risks, as well as some procedures requiring OLV and lateral positioning and their associated complications. Harlequin syndrome is a unique complication to MICS.

What is 'off-pump' surgery?

'Off-pump' cardiac surgery does not employ cardiopulmonary bypass and cardioplegia, thus avoiding the potential systemic inflammatory response, coagulopathy and transfusion requirements, and potential for renal or neurological injuries. It can facilitate shorter ICU and hospital stays. The same preparations, monitoring and anaesthetic considerations are required, as well as excellent communication between anaesthetist and surgeon.

What are potential complications of 'off-pump surgery'?

There are specific complications the anaesthetist should be aware of. Cardiovascular instability and cardiac ischaemia are more common. To obtain local wall stabilisation, cardiac lifting, repositioning and retractors are often required. Stabilisers or shunts may also be applied. These can all lead to decreases in preload, afterload and stroke volume. Such changes can cause a reduction in coronary vessel filling and thus ischaemia or cardiovascular compromise, including profound hypotension and arrhythmias. Management includes maintaining a MAP of greater than 70 mmHg with a combination of fluid boluses, vasopressors or Trendelenburg position. Any arrhythmias should be treated aggressively, considering β -blockers, magnesium or cardioversion. Close discussion with the surgeon is key and conversion to CPB may be required.

Temperature management can be difficult during 'off-pump' procedures with significant heat loss due to the open thorax. CPB is frequently used to cool and rewarm patients. Without it, non-invasive methods are relied upon. Once a patient has become hypothermic, it can be difficult to effectively rewarm them prior to the end of the operation.

Patients may still require anticoagulation with heparin at a reduced dose, and reversal with protamine, encountering the same complications.

What is ERAS Cardiac and how does it differ from standard treatment of patients undergoing cardiac procedures?

Again, keep it simple, in-depth knowledge is not required. An overview of the basic principles should suffice.

ERAS Cardiac is the Enhanced Recovery After Cardiac Surgery perioperative initiative. It aims to standardise care to expedite patient recovery and reduce complications. It may start prior to admission with 'prehabilitation', including review of exercise, diet and smoking-cessation advice to optimise cardiovascular and respiratory reserve.

Preoperatively, minimal starvation times and preoperative oral carbohydrate loading are advocated. Meticulous attention should be given to infection control measures, reducing the risk of surgical site infections. Throughout the perioperative period, glycaemic control should be monitored and maintained where necessary.

Postoperatively, patients should be extubated within 6 hours, hypothermia actively avoided, and multimodal opioid-sparing analgesia initiated to allow early mobilisation. Early removal of drains and catheters should be considered where appropriate and patients screened daily for delirium.

What antibiotic strategies are used to reduce infection in cardiac surgery?

Postoperative infections are associated with a significant increase in morbidity and mortality. Patients are screened preoperatively for staphylococcal colonisation, and treatment given where required, usually via intranasal ointment and skin wash. Intraoperatively, alongside meticulous sterility, appropriate prophylactic intravenous antibiotics should be given and continued as per local policies.

Further Reading

Jameel S, Colah S, Klein AA. Recent advances in cardiopulmonary bypass techniques. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2010; 10(1): 20–23.

Parnell A, Prince M. Anaesthesia for minimally invasive cardiac surgery. *British Journal of Anaesthesia Education*. 2018; 18(10): 323–330.

Pokhrel S, Gregory A, Mellor A. Perioperative care in cardiac surgery. *British Journal of Anaesthesia Education*. 2021; 21(10): 396–402.

Sannakki S, Sannakki D, Echebarria J, Patterial M. Preoperative assessment for cardiac surgery. *Anaesthesia and Intensive Care Medicine, Cardiac Anaesthesia*. 2021; 22(4): 216–222.

2.1.2 Anaesthesia of the Cardiac Patient for Non-cardiac Surgery – Michael John Scerri and Andrea Binks

A 70-year-old man presents to the pre admissions clinic in preparation for a colonic resection for an invasive sigmoid tumour. He had an acute myocardial infarction 10 years ago managed with coronary artery bypass grafting. He has stable angina on moderate exertion and a recent coronary angiogram showed further significant single vessel coronary artery disease, not amenable to surgical or percutaneous treatment. His other significant comorbidities include hypercholesterolaemia, hypertension and atrial fibrillation for which he is currently taking rosuvastatin, perindopril, metoprolol and rivaroxaban.

What are the main issues with this man?

You need to summarise the salient points, always considering context. Why is this patient presenting at this point in time, for this surgery? How urgent is this surgery and how will the stability and severity of any acute or chronic comorbidities impact his perioperative journey?

This case presents an elderly man requiring a time sensitive surgical procedure for an invasive colonic cancer, which may have devastating complications if not treated with haste. He has numerous comorbidities which will increase his perioperative risk including ischaemic heart disease, hypertension and atrial fibrillation, for which he is anticoagulated. The acute physiological impact of his invasive cancer and the severity and stability of these chronic comorbidities and their impact on his functional capacity must be assessed and a decision on the balance of risk versus benefit of proceeding with such a case made.

What are the goals of your assessment of this patient in the pre-admissions clinic?

Practise a structure to ensure you are thorough but also swift in your response to such an open-ended question.

The goals of my assessment in the pre-admissions clinic are to assess the patient's perioperative risk for this surgery, identify the impact his comorbidities will place on his perioperative course, and identify which comorbidities require optimisation preoperatively. I would take the opportunity to formulate a perioperative plan including post-operative disposition and ensure these are discussed with the patient, his family and care givers. Time should be provided for clarity, any questions answered and informed consent for anaesthesia and any procedures to be performed obtained.

What would your preoperative assessment focus on?

My preoperative assessment would entail a history, examination and collation of investigations focussed on the patient's acute primary disease and to determine the severity and stability of his comorbid illnesses.

Regarding the acute pathology, I would establish a timeframe of disease progression and clarify disease staging. I would consider the mass effect of the tumour on bowel motility, obstruction and aspiration risk, as well as the mass effect on extracolonic organs. It would be important to elicit the presence of any extracolonic metastases, with particular focus on hepatic, respiratory or intracranial. Assessment for the presence of pleural effusions, restrictive lung disease, hepatic dysfunction, raised intracranial pressure and altered cognition should be performed. The metabolic effect of the tumour should be considered, along with the presence of any paraneoplastic syndromes; for example, secondary to a neuroendocrine tumour or carcinoid syndrome. In the presence of such syndromes, the role of suppressive agents such as octreotide, should be considered. Fluid and electrolyte balance and nutritional status should be assessed. The presence of anaemia will be particularly important to establish and manage in the context of ischaemic heart disease, stable angina and atrial fibrillation.

The severity and stability of his chronic illnesses should be assessed. This patient appears to have stable angina and the metabolic equivalents (METs) at which the patient can function needs to be established. Regarding atrial fibrillation, confirmation of adequate rate and or rhythm control is imperative as is questioning for a history of embolic phenomenon, particularly any prior cerebrovascular accidents. I would calculate a CHADsVasc score to estimate risk of such events and to help guide my perioperative anticoagulation plan. After a focussed assessment, I would conclude with a systems review, previous issues with anaesthesia, the presence of any drug reactions or allergies and finally an assessment of the patient's airway. To conclude, review of any pre-completed bedside, laboratory, imaging or other relevant available investigations would be performed.

What will guide your decision on investigations and what investigations would you consider?

My preoperative investigations will be guided by my assessment of the patient and consideration of the surgery that is planned. I would follow any local hospital policies and consider endorsed guidelines regarding the appropriateness of perioperative investigations, ensuring any performed may have the potential to subsequently alter the patient's perioperative management.

Bedside investigations would include the patient's weight, height, 12-lead ECG and a blood sugar measurement. Laboratory investigations would include a full blood count, particularly characterising any anaemia, iron studies, renal function, electrolyte

abnormalities, liver function tests and coagulation profile. A group and screen should be organised closer to the date of surgery so it remains valid during the procedure and postoperatively. I would review any imaging such as a chest X-ray, CT scan, echocardiography or cardiologist reports if available. If the presence of carcinoid syndrome is suspected, consideration of a subsequent thorough cardiovascular workup including repeated echocardiography should be performed to exclude right-sided cardiac disease. Regarding cardiac investigations, they should mirror standard practice in the non-operative setting. If the patient's functional capacity was unable to be accurately assessed, formal objective stress testing should be considered, in either the form of cardiopulmonary exercise stress testing or non-invasive testing assessing for the presence of fixed or reversible myocardial lesions.

How would you assess his functional capacity and what is the importance of this?

Functional capacity can be described by a term called METS (metabolic equivalents), with one MET representing basal metabolic rate. The gold standard objective assessment of METS would include cardiopulmonary exercise testing (CPET), though this is not often suitable in patients with physical limitations unable to reach their target heart rate. Poor performance in CPET has been correlated with significantly increased adverse perioperative cardiac events, while the ability to reach high workloads is associated with only minor increases.

Metabolic equivalents can otherwise be assessed by history, utilising a tool such as the Duke Activity Status Index (DASI). The ability of a patient to take care of themselves, walk indoors or 100 metres on level ground at 5 km/hour would represent one to four METS, with the ability to climb two flights of stairs approximating four METS. Although the preoperative assessment of METS has shown a weak correlation to postoperative cardiac complications, it is notable that even in the presence of cardiac disease, when a patient's functional capacity is excellent, the prognosis is usually excellent. The 2014 ESA / ESC guidelines for perioperative assessment states level one evidence supporting the use of imaging stress testing in patients undergoing high-risk surgery with a functional capacity less than four METS and three or more clinical risk factors as described by Lee's Revised Cardiac Risk Index.

What factors in this case would place this patient at a heightened risk for cardiac complications?

Risk factors for perioperative cardiac events are influenced by patient, surgical and situational factors. Patient-specific factors appear to be the main predictor of perioperative cardiac complications, but procedure and anaesthetic factors cannot be ignored. Multiple risk indices have been developed, with Lee's Revised Cardiac Risk Index being very well known and considered by many clinicians as the best risk stratification score to predict major adverse cardiac events in non-cardiac surgery. The revised index includes six variables: surgery type, history of ischaemic heart disease, cerebrovascular disease, heart failure, preoperative creatinine levels and the use of insulin to manage diabetes mellitus. This case involves an urgent intermediate risk surgery, likely of prolonged duration, with potential for significant fluid, electrolyte and core temperature

fluctuations. Significant tissue injury and an incitement of a high level of surgical stress would be expected, all identified as risk factors for perioperative cardiac adverse events. This stress can lead to an imbalance in myocardial oxygen supply and demand and also alter the balance of prothrombotic and fibrinolytic processes, potentially increasing coronary thrombogenicity.

Are there any scoring systems you could use to estimate this risk?

Lees Revised Cardiac Risk Index, although used widely, may be relatively dated. A new National Surgical Quality Improvement Programme (NSQIP) scoring system was developed and validated in 2008, providing a model-based estimate of IHD or cardiac arrest within 30 days for an individual patient. The NSQIP cardiac risk calculator identified five variables (type of surgery, functional status, elevated creatinine, ASA class and age) and outperformed Lee's Revised Index for the primary outcome. One downfall, however, is Lee's Revised Cardiac Index's inclusion of other clinically significant outcomes, such as pulmonary oedema and complete heart block, which are not addressed in the NSQIP score. Lee's Revised Index performed moderately well in patients at low and high risk, but performed poorer in vascular procedures and when predicting death. It is therefore suggested these two tools are utilised in a complementary manner throughout the decision-making process.

Can you outline the use of cardiac biomarkers in predicting cardiac risk in non-cardiac surgery?

B-type natriuretic peptide (BNP) and N terminal pro BNP (NT-ProBNP) are peptides produced by the myocardium during episodes of stress, including heart failure in the absence of myocardial infarction. In the setting of non-cardiac vascular surgery, BNP and NT-ProBNP have been successfully used to prognosticate cardiac events and mortality. However, evidence of their effectiveness in other surgical settings is less robust, with routine use not recommended. The likelihood of a benefit of these tests lies in patients who are already identified as high risk, as described by poor functional status or a Lee's Revised Cardiac Risk score of three or more.

This patient is on rivaroxaban for atrial fibrillation. What will be your perioperative anticoagulation plan?

Management of perioperative anticoagulation should be based on patient, surgical and anaesthetic factors. The risk-benefit ratio of ceasing such medications preoperatively needs to be assessed, as does the requirement for bridging anticoagulation therapy with heparin or low molecular weight heparin. The bleeding risk of the surgery proposed needs to be considered against the thrombotic risk in the perioperative period. Consideration must also be given to the plan for invasive anaesthetic procedures, such as neuraxial regional anaesthesia, which will require a period of abstinence from anticoagulants in their own right. The timing of such decisions depends on the pharmacokinetic and pharmacodynamic profile of each anticoagulant and any metabolic or excretory abnormalities of the patient, namely the presence of renal or hepatic dysfunction.

Regarding this patient, I would consider this surgery high risk for bleeding and part of my anaesthesia plan will likely involve a neuraxial technique. Non vitamin

K antagonist oral anticoagulants (NOACS) have a more predictable pharmacokinetic profile, and in the absence of risk factors for a high risk of thrombus formation, cessation usually does not warrant bridging. Rivaroxaban clearance is less dependent on renal excretion (33%) with a median half-life of 12 hours. Ideally cessation of rivaroxaban in this context should be four to five times the biological half-life, and therefore cessation for 48–72 hours should suffice. Importantly, an anticoagulation recommencement plan in the postoperative period should be discussed with the surgical team, with restarting of rivaroxaban therapy usually delayed 24–48 hours post-surgery.

How will you perform your anaesthetic for this case?

Having a mnemonic will help you in your explanation of how you would prepare and perform your anaesthetic. Practise these if you think they will help.

I would first ensure the case is indicated, there are no contraindications to proceeding and the patient is understanding of and consenting to the procedures discussed. I would then consider the position required for the surgery and arrange my anaesthetic equipment accordingly. With standard AoA monitoring in place, specifically a 5-lead electrocardiogram to detect intraoperative ischaemia and a radial arterial line to monitor haemodynamics closely, I would place a large gauge intravenous canula and a central venous catheter, to help guide fluid management and administer vasopressors and inotropes as required.

If the procedure is planned to be open, I would place a thoracic epidural to provide intra- and postoperative analgesia, with the goal of reducing the surgical stress response and its adverse effects on the balance of myocardial oxygen supply and demand. The 2018 Enhanced Recovery After Surgery (ERAS) guidelines specifically focussed on colorectal surgery advocates for epidural analgesia as the gold standard for postoperative analgesia in open colorectal surgery. Comparatively a thoracic epidural could be placed for minimally invasive or laparoscopic surgery; however, given the equianalgesic effect of a single shot intrathecal technique and abdominal trunk blocks in minimally invasive surgery, epidural insertion cannot be advocated for in place of a multimodal technique. The abdominal trunk block of choice would be to place bilateral transversus abdominis plane (TAP) catheters, with a continued infusion of local anaesthetic in the postoperative phase for analgesia.

I would perform a total intravenous anaesthesia technique with propofol, placing a bispectral index monitor to monitor depth of anaesthesia. The patient will be intubated with a standard endotracheal tube and I would aim to utilise opioids to blunt the sympathetic response to intubation, depending on the epidural to provide intraoperative analgesia. Importantly a vasopressor would be administered concurrently during the induction and maintenance phases, with either metaraminol or noradrenaline titrated to maintain a suitable mean arterial pressure.

I would remain vigilant with antibiotic prophylaxis, temperature, fluid, electrolyte and blood glucose management. Upper and lower body warmers, along with a fluid warmer should be utilised to minimise heat loss. Given the procedure duration and position likely to be steep head down, I would maintain strict pressure area care and consider airway swelling prior to extubation. My goal would be to extubate this patient postoperatively, and after a period of postoperative recovery, be electively transferred to the high dependency unit. Postoperatively, specific planning around recommencement of regular medications, in particular anticoagulants, and timing of the removal of the epidural needs to be communicated.

Further Reading

- Camm J, Lip G, De Caterina R, Savelieva I, Atar D, Hohnloser S, Hendricks G, Kirchhof P. 2012 focused update of the ESC guidelines for the management of atrial fibrillation. *European Heart Journal*. 2012; 33; 2719–2747.
- Fleisher LA, Fleischmann KE, Auerbach AD, Barnason SA, Beckman JA, Bozkurt B, Davila-Roman VG, Gerhard-Herman MD, Holly TA, Kane GC, Marine JE, Nelson MT, Spencer CC, Thompson A, Ting HH, Uretsky BF, Wijeysondera DN. 2014 ACC/AHA guideline on perioperative cardiovascular evaluation and management of patients undergoing noncardiac surgery: A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *Circulation*. 2014; 130, e278–e333.
- Ford MK, Beattie WS, Wijeysondera DN. Systematic review: Prediction of perioperative cardiac complications and mortality by the revised cardiac risk index. *Annals of Internal Medicine*. 2010; 152, 26–35.
- Gustafsson UO, Scott MJ, Hubner M, Nygren J, Demartines N, Francis N, Rockall TA, Young-Fadok TM, Hill AG, Soop M, de Boer HD, Urman RD, Chang J, Fichera A, Kessler H, Grass F, Whang EE, Fawcett WJ, Carli F, Lobo DN, Rollins KE, Balfour A, Baldini G, Reidel B, Ljungqvist O. Guidelines for perioperative care in elective colorectal surgery: Enhanced Recovery After Surgery (ERAS) Society recommendations: 2018. *World Journal of Surgery*. 2019; 43, 659–695.
- Karthikeyan G, Moncur RA, Levine O, Heels-Ansdell D, Chan MT, Alonso-Coello P et al. Is a pre-operative brain natriuretic peptide or N-terminal pro-B-type natriuretic peptide measurement an independent predictor of adverse cardiovascular outcomes within 30 days of noncardiac surgery? A systematic review and meta-analysis of observational studies. *Journal of the American College of Cardiology*. 2009; 54, 1599–1606.
- Mangano DT. Peri-operative medicine: NHLBI working group deliberations and recommendations. *Journal of Cardiothoracic and Vascular Anaesthesia*. 2004; 18, 1–6.
- Morris CK, Ueshima K, Kawaguchi T, Hideg A, Froelicher VF. The prognostic value of exercise capacity: A review of the literature. *American Heart Journal*. 1991; 122, 1423–1431.
- Powell B, Al Muhktar A, Mills G. Carcinoid: The disease and its implications for anaesthesia. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11(1), 9–13.
- The Joint Task Force on non-cardiac surgery: Cardiovascular assessment and management of the European Society of Cardiology (ESC) and the European Society of Anaesthesiology (ESA) 2014 ESC/ECA Guidelines on non-cardiac surgery: Cardiovascular assessment and management. *European Heart Journal*. 2014; 35, 2383–2431.
- Wiklund RA, Stein HD, Rosenbaum SH. Activities of daily living and cardiovascular complications following elective, noncardiac surgery. *Yale Journal of Biological Medicine*. 2001; 74, 75–87.

2.1.3 Congenital Heart Disease – Luke Foster

Tell me about congenital heart disease.

Congenital heart disease may refer to any one of a number of structural abnormalities of the heart or intrathoracic great vessels that is present at birth and is, or may become, of functional significance. It results from a failure of normal development of the circulatory system and is the most common form of live birth defect, affecting approximately 1 in 100 live births.

Risk factors for the development of congenital heart disease include advanced maternal age, family history of congenital heart disease, maternal smoking, maternal autoimmune disease, maternal infections including rubella, and exposure to toxins and teratogenic medication. There are several genetic syndromes associated with congenital heart disease, which include Down's and Turner's syndrome.

Just over half of patients will be diagnosed in infancy, with most of the remainder diagnosed in childhood and approximately 10% diagnosed in adulthood. More than 90% of children with CHD survive into adulthood and the prevalence of adults with Grown Up Congenital Heart Disease (GUCH) in the community now outnumbers the number of children with congenital heart disease.

How would you classify congenital heart disease?

Congenital heart disease can be classified according to complexity, haemodynamic abnormality, presence or absence of cyanosis, or duct-dependence. The European Society of Cardiology and American Heart Association classifications rank congenital lesions by their complexity into either mild, moderate or severe (see Table 2.1.3.1). An alternative system based on the haemodynamic and clinical consequences classifies the resultant circulation as either in series (normal), in parallel (balanced) or based around a single ventricle. These circulations may be encountered if the patient has undergone palliative or corrective surgery, or has had no intervention performed. See Figure 2.1.3 for schematic diagrams of examples of these.

In a series or normal circulation, there are separate systemic and pulmonary circulations working together in series. Congenital heart disease in series circulations may take the form of simple obstructive lesions, for example aortic stenosis or coarctation of the aorta, simple left-to-right shunt lesions causing increased pulmonary blood flow, for example a ventricular septal defect (VSD) or patent ductus arteriosus (PDA), or simple right-to-left shunt lesions causing a reduction in pulmonary blood flow and cyanosis, for example the tetralogy of Fallot. Achieving a series circulation is the aim for most corrective surgical procedures.

In a parallel or balanced circulation, the pulmonary and systemic circulations are in communication with one another, and function as if in parallel. The proportion of the total blood flow received in either the systemic or pulmonary circulation depends on the relative resistance in each circuit. It is said to be a 'balance' of the systemic and pulmonary vascular resistance. Excessive blood flow to the pulmonary circulation results in pulmonary congestion, poor systemic perfusion and, if prolonged, pulmonary hypertension. Inadequate blood flow to the pulmonary circulation results in cyanosis. Examples include a large unrepaired atrio-ventricular septal defect or transposition of the great arteries.

In a single ventricle circulation, there is a single functional ventricle which pumps blood to both the systemic and pulmonary circulations. These circulations can be classified according to whether or not the pulmonary and systemic venous blood is either mixed, resulting in chronic cyanosis, or separated, with the systemic venous blood connected directly to the pulmonary artery. This is the Fontan circulation. A single ventricle circulation may be the consequence of a congenital lesion, for example, double inlet left ventricle, or the final result of staged palliative surgical procedures in those with complex congenital heart disease in whom a biventricular repair is not possible. The

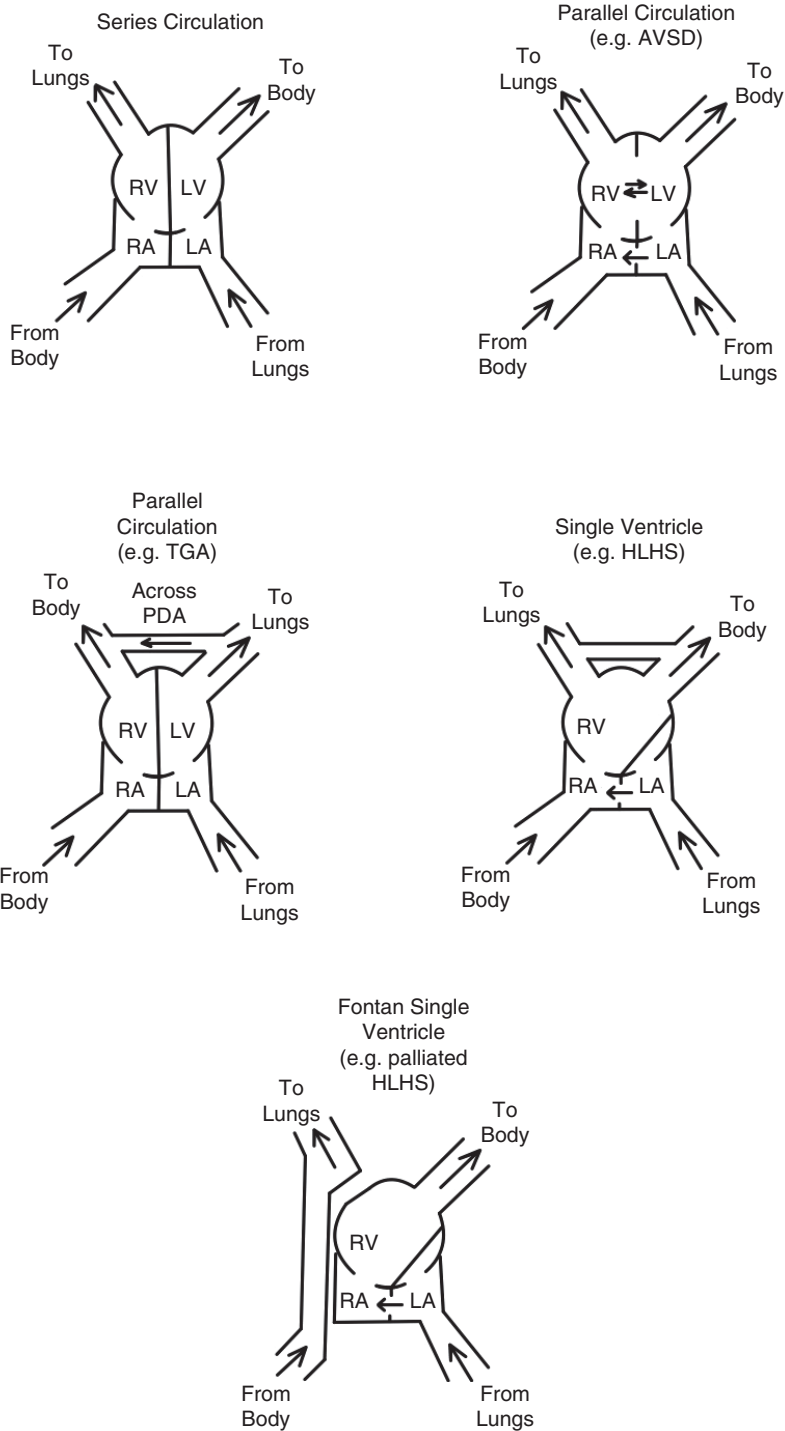


Figure 2.1.3 Schematic diagrams of a normal circulation, parallel circulations resulting from congenital heart disease and one based on a single ventricle before and after surgery.

Table 2.1.3.1 Classification of congenital heart disease by complexity

Simple lesion	Isolated aortic valve disease and bicuspid aortic disease Isolated mitral valve disease Mild isolated pulmonary stenosis Isolated small ASD, VSD or PDA Repaired secundum ASD, sinus venosus defect, VSD or PDA without residual defect or sequelae
Moderate complexity	Subvalvular or supravalvular aortic stenosis AVSD, partial or complete Repaired Tetralogy of Fallot Coarctation of the aorta Ebstein anomaly TGA after arterial switch repair PDA, moderate or large unrepaired VSD with associated abnormalities and/or moderate or greater shunt
Severe	Any repaired or unrepaired congenital heart disease with pulmonary vascular disease including Eisenmenger syndrome Any cyanotic congenital heart disease (unoperated or palliated) Fontan circulation Pulmonary atresia Single ventricle circulation

systemic ventricle may be morphologically either a right or left ventricle, which carries prognostic significance in terms of its ability to cope with the workload required.

In the Fontan circulation, blood flow in the pulmonary circulation is passive, and is therefore dependent on the transpulmonary gradient: the pressure gradient between the pulmonary artery and the left atrium. The significance of passive pulmonary blood flow is that any factor increasing either pulmonary vascular resistance or intrathoracic pressure will cause a reduction in both pulmonary perfusion as well as cardiac output.

An alternative classification system describes lesions as either cyanotic or acyanotic. For a lesion to be acyanotic, it must have either no communication between the systemic and pulmonary circulations (e.g., aortic stenosis), or a communication with a dominant left-to-right shunt between them (e.g., a ventricular or atrial septal defect). Cyanotic lesions require a significant amount of mixing of blood between the pulmonary and systemic circulations, and may be present initially, or the result of reversed blood flow across a previously left-to-right shunt, i.e., Eisenmenger’s syndrome.

What are the consequences of cyanotic congenital heart disease?

Chronic cyanosis has multiple systemic implications including impaired growth and development, polycythaemia, thromboembolic disease including paradoxical embolism, increased bleeding risk, chronic kidney disease with impaired urate clearance resulting in gout and kidney stones, pigment gallstones and an increased risk of brain abscesses.

How might a child with congenital heart disease present?

In children not diagnosed antenatally, suggestive symptoms include a history of difficulty with feeding or sweating while feeding, tachypnoea, grunting, and cyanotic spells

precipitated by feeding or crying. Clinical signs may include the presence of a pathological murmur, hepatomegaly, oedema or radio-femoral delay.

During the transition from the fetal circulation, the ductus arteriosus functionally closes within the first 96 hours of life. Some children require a patent duct in order to perfuse either the pulmonary or systemic circulations. This is referred to as duct-dependence. If the duct is required to perfuse the pulmonary circulation (e.g., tricuspid atresia) this may result in cyanosis when it closes. If it is required to perfuse the systemic circulation (e.g., hypoplastic left heart syndrome), then closure may result in systemic hypoperfusion and cardiorespiratory collapse.

Duct-dependent congenital heart disease should be high on the list of differential diagnoses in an infant presenting with sudden unexpected postnatal collapse. Suggestive clinical features include cyanosis, hypoxia despite oxygen therapy, persistent lactic acidosis unresponsive to initial resuscitation, tachypnoea in the absence of overt lung pathology, absent femoral pulses and clinical evidence of heart failure. Other important diagnoses to consider include sepsis, metabolic disease and non-accidental injury.

Specific management of this neonatal emergency is with diagnostic echocardiography and an infusion of alprostadil, a prostaglandin E1 analogue, to maintain duct patency. Supportive treatment includes airway management due to alprostadil's tendency to cause apnoeic episodes, and the manipulation of pulmonary and systemic vascular resistance to maintain tissue oxygenation and perfusion. The measurement of pre-ductal oxygen saturations on the right hand, and post-ductal saturations on either foot should target oxygen saturations of 75–85%. Higher oxygen saturations can lower the pulmonary vascular resistance and increase pulmonary blood flow at the expense of systemic perfusion, resulting in a pink but profoundly shocked child. Hypotension should be avoided as this results in increased right-to-left shunt and worsening cyanosis.

What are the specific considerations when anaesthetising a patient with congenital heart disease?

Improvements in life expectancy for children with congenital heart disease has led to the increasing incidence of adults with Grown Up Congenital Heart Disease. It is therefore increasingly common for patients with congenital heart disease to present for either elective or emergency surgery to non-specialist centres.

The European Society for Cardiology's 2020 guidelines for the management of adult congenital heart disease identify patient, medical and surgical factors associated with increased risk of perioperative morbidity and mortality in those undergoing non-cardiac surgery. Patient factors include younger age and the presence of complex congenital heart disease. Medical factors include the presence of heart failure, cyanosis, pulmonary hypertension, and poor general health. Surgical factors include operations performed on the respiratory or nervous systems, and urgent or emergency surgery.

The anaesthetic assessment of a patient with congenital heart disease should seek to understand the complexity of the congenital lesion, the physiological status of the patient, and the complexity and nature of the proposed surgery. Complex congenital lesions are defined as those with single ventricle physiology, parallel circulation physiology resulting in cyanosis, cardiomyopathy and severe or symptomatic aortic stenosis. Assessing the degree of patient compensation for their lesion is important, and symptoms or signs that confer an elevated risk include cardiac failure, pulmonary hypertension, cyanosis and arrhythmias. The finding of ventricular ectopics on a 12-lead ECG in

children is an ominous sign. Major surgery is defined as intraperitoneal or intrathoracic surgery, or any surgery where major blood loss requiring transfusion is anticipated. High-risk procedures therefore are those where poorly compensated patients with complex congenital lesions require major emergency surgery. These patients should be transferred emergently to a specialist centre where possible. Low-risk procedures will include minor elective surgery performed on well-compensated patients with simple congenital lesions, and these will not necessarily require specialist input.

The anaesthetic assessment should also consider and address issues including the presence of associated congenital conditions, for example, Down's, Turner's or Di George syndrome, the presence of additional comorbidities including ischaemic heart disease, diabetes and hypertension, the potential for difficult venous or arterial access, the risk of associated arrhythmias and need to manage the patient's pacemaker or implantable cardiac defibrillator, the management of perioperative anticoagulation and bleeding risk, consideration for antibiotic prophylaxis to prevent endocarditis and the strict prevention of air emboli in venous lines to prevent paradoxical cerebral emboli in those with persistent shunts.

Following this approach, it is evident that many possible scenarios can be encountered, ranging from a 6-year-old with a repaired ventricular septal defect presenting for a dental extraction, to a 21-year-old with decompensated single ventricle physiology and chronic cyanosis requiring an emergency endoscopy for an upper GI bleed!

The European Society for Cardiology's 2020 guidelines include a modified version of the World Health Organisation classification of maternal cardiovascular risk to include congenital diseases. This serves as a useful guide upon which to estimate risk. In this classification, patients in WHO risk class III and IV will require management in a specialist centre, while those in class I and II generally will not. Those in class II and III should usually be discussed for consideration of transfer to a specialist unit. See Table 2.1.3.2 for the list of conditions and their risk classification.

Which groups of patients should receive antibiotic prophylaxis against infective endocarditis?

Guidance on antibiotic prophylaxis for infective endocarditis is conflicting. The UK NICE clinical guideline recommends against routine prophylaxis for any procedure, but states that patients at risk of infective endocarditis may require antibiotic therapy that covers organisms causing infective endocarditis when invasive gastrointestinal or genitourinary procedures are performed at a site of suspected infection. Antibiotic therapy is needed when invasive procedures are performed in the context of infection.

The European Society for Cardiology takes a different approach, and maintains the principle of antibiotic prophylaxis in patients deemed at highest risk undergoing the highest risk dental procedures only. High-risk patient groups include those with prosthetic valves or where prosthetic material was used for a valve repair, any previous episode of infective endocarditis, any cyanotic congenital heart disease, and for six months in those who have undergone any congenital repair where prosthetic material was used or lifelong if there is persistent shunt or valvular regurgitation. The main targets of antibiotic regimes are oral streptococci and so amoxicillin, cephalosporins or clindamycin are commonly recommended.

Table 2.1.3.2 European Society of Cardiology 2018 Modified WHO classification of maternal cardiovascular risk including congenital lesions. Reproduced with permission from Oxford University Press.

mWHO risk class	Diagnosis
I	<p><i>Uncomplicated or small:</i></p> <ul style="list-style-type: none"> – Pulmonary stenosis – Patent ductus arteriosus – Mitral valve prolapse <p><i>Successfully repaired simple:</i></p> <ul style="list-style-type: none"> – Atrial or ventricular septal defect – Patent ductus arteriosus – Anomalous pulmonary venous drainage – Atrial or ventricular ectopic beats, isolated
II	<p><i>If otherwise well and uncomplicated:</i></p> <ul style="list-style-type: none"> – Unoperated atrial septal defect or ventricular septal defect – Repaired Tetralogy of Fallot – Most arrhythmias (SVT) – Turner syndrome without aortic dilatation
II–III	<p>Repaired coarctation</p> <p>Atrioventricular septal defect</p> <p>Native or tissue valvular heart disease not considered WHO I or IV (mild mitral stenosis, moderate aortic stenosis)</p> <p>Aorta <45 mm in bicuspid aortic valve pathology</p>
III	<p>Systemic right ventricle with good or mildly decreased ventricular function</p> <p>Fontan circulation if otherwise well and uncomplicated cardiac condition</p> <p>Unrepaired cyanotic heart disease</p> <p>Other complex congenital heart disease</p> <p>Moderate mitral stenosis</p> <p>Severe asymptomatic aortic stenosis</p> <p>Moderate aortic dilatation</p> <p>Ventricular tachycardia</p>
IV	<p>Pulmonary artery hypertension of any cause</p> <p>Severe mitral stenosis</p> <p>Severe symptomatic aortic stenosis</p> <p>Systemic right ventricle with moderate or severely decreased ventricular function</p> <p>Severe aortic dilatation</p> <p>Severe (re)coarctation</p> <p>Fontan with any complication</p>

Further Reading

Baumgartner H, De Backer J, Babu-Narayan SV et al. 2020 ESC guidelines for the management of adult congenital heart disease. *European Heart Journal*. 2021; 42, 563–645.

Flannery KM, Raviraj D. Anaesthesia in children with congenital heart disease for noncardiac surgery *Anaesthesia Tutorial of the Week Tutorial* 467. March 2022.

Greaney D, Honjo O, O’Leary JD. The single ventricle pathway in paediatrics for

- anaesthetists. *BJA Education*. 2019; 19(5): 144–150.
- Lal N, Varshney T. The collapsed newborn in the emergency department *BJA Education*. 2018; 18(8): 254–258.
- Liu Y et al. Global birth prevalence of congenital heart defects 1970–2017: Updated systematic review and meta-analysis of 260 studies. *International Journal of Epidemiology*. 2019; 48(2): 455–463.
- White MC, Peyton JM. Anaesthetic management of children with congenital heart disease for non-cardiac surgery. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2012; (12)1: 17–22.
- 2018 ESC guidelines for the management of cardiovascular diseases during pregnancy. *European Heart Journal*. 2018; 39: 3165–3241.
- 2015 guidelines for the management of infective endocarditis *European Heart Journal*. 2015; 36, 3075–3123.
- National Institute for Health and Care Excellence (NICE) Clinical guideline CG64 Prophylaxis against infective endocarditis: Antimicrobial prophylaxis against infective endocarditis in adults and children undergoing interventional procedures – last updated July 2016.

2.1.4 Care of the Postoperative Cardiac Surgery Patient – Luke Foster

What complications of cardiopulmonary bypass are seen in the ICU?

Cardiopulmonary bypass triggers a major systemic inflammatory response with resultant multiorgan dysfunction, mimicking that seen in sepsis or trauma. Pro-inflammatory cytokine release and activation of complement alongside the coagulation and fibrinolysis cascades results in increased oxygen consumption, reduced systemic vascular resistance, increased capillary permeability, endothelial dysfunction, coagulopathy, thrombosis and end organ damage. Key triggers include exposure of the patient's blood to non-pulsatile flow within an extracorporeal circuit, the reperfusion of ischaemic myocardium, multiple blood product transfusions, haemodilution and alterations in body temperature. The consequences are seen in the intensive care unit, and the duration of cardiopulmonary bypass can indicate the degree to which postoperative physiology is deranged, alongside other patient, anaesthetic and surgical factors.

What cardiac complications are there?

Cardiac complications are numerous and a degree of myocardial dysfunction is expected coming off bypass. Postoperative low cardiac output state is common, and diastolic dysfunction and myocardial stunning are the result of myocardial oedema, cardiectomy and low-grade myocardial injury. Frank myocardial ischaemia is less common but may be the consequence of graft failure, air embolism or inadequate cardioplegia delivery. Tachy- and bradyarrhythmias may result from electrolyte disturbance, hypothermia, direct conduction system trauma or myocardial oedema. The right ventricle may be particularly vulnerable to dysfunction as it is poorly tolerant of volume overload and increased pulmonary vascular resistance. Finally, cardiac tamponade is a common and important cause of low cardiac output state.

Tell us more about postoperative bleeding.

Postoperative bleeding is common following cardiac surgery and can be divided into surgical and medical causes. Surgical causes include inadequate haemostasis, vessel

injury and graft anastomosis failure. Medical causes result from the systemic inflammatory response to cardiopulmonary bypass as well as the residual effects of heparin and antiplatelet medication. Platelet dysfunction, coagulation factor depletion and excessive fibrinolysis can all be responsible and contributing factors include haemodilution, hypothermia, hypocalcaemia, postoperative hypertension and the short half-life of protamine. Excessive fibrinolysis is routinely pre-emptively treated with tranexamic acid, which has been shown to reduce blood transfusion requirements.

The definition of significant postoperative bleeding varies but, in general, drain outputs of more than 400 ml/hr over the first hour, 200 ml/hr for two consecutive hours or 100 ml/hr for four consecutive hours warrants surgical re-exploration in the absence of a correctable medical cause. Point-of-care testing with thromboelastography (TEG) or platelet function assays are useful to identify the cause of bleeding and guide appropriate therapy. Residual heparin effect can be reversed with protamine, platelet dysfunction may require treatment either with desmopressin or platelet transfusion, and fresh frozen plasma or cryoprecipitate can replace clotting factors and fibrinogen respectively. Transfusion of red blood cells is generally indicated at haemoglobin levels below 70 g/L although some units may aim to maintain levels higher than this.

Do you anticipate any respiratory complications?

Respiratory complications are attributable to the impact of median sternotomy on pulmonary mechanics. Reduced chest wall compliance contributes to a marked reduction in lung volumes that can persist for weeks following surgery. Phrenic nerve injury can be caused by surgical retractors, topical cardiac cooling or direct trauma in the context of difficult dissection and may result in an elevated hemi-diaphragm on the affected side. Increased lung water as a result of haemodilution, poor left ventricular function, fluid overload and the systemic inflammatory response to bypass is also common. These factors all contribute towards ventilatory/perfusion mismatch and hypoxaemia.

What neurological complications of cardiopulmonary bypass are there?

Neurological complications following bypass include those affecting the central and peripheral nervous systems. Peripheral nerve injuries include brachial plexus injury from crucifix positioning and surgical traction, anterior intercostal nerve injury sustained while harvesting the internal mammary artery, and phrenic nerve palsy which can arise from direct cutting of the nerve in the case of difficult dissection, excessive retraction or neuropraxia resulting from the use of topical cardiac cooling with ice.

Post-bypass central nervous system events can be divided into type 1 neurological injury, which encompasses focal stroke and TIA, and type 2 neurological injury which describes a more diffuse or global injury with cognitive decline. Cerebrovascular ischaemia is both an important neurological complication and predictor of postoperative mortality. The reported incidence of clinically apparent stroke following isolated coronary artery bypass surgery is 1.6%, but it may be up to 13% after open chamber, for example, valve surgery. Postoperative cognitive dysfunction is significantly more common, with short-term (i.e. less than six weeks) cognitive dysfunction occurring in up to a half of patients, and longer term cognitive dysfunction persisting at six months in up to a third.

Patient risk factors for the two conditions overlap, and include advanced patient age, carotid stenosis, previous cerebrovascular disease, aortic atheroma, pre-existing poor

ventricular function and diabetes. Procedural aetiological factors may include particulate or gaseous emboli, cerebral hypoperfusion, pH-stat pH management (with associated loss of cerebral autoregulation), long bypass times, excessive haemodilution, systemic inflammatory response, hyperglycaemia and poor temperature control.

Current evidence-based recommendations for the prevention of CNS injury following bypass include the use of aortic ultrasound to identify non-palpable aortic atheroma, preoperative carotid ultrasound to identify carotid stenosis, the incorporation of arterial line filters in the bypass circuit to minimise the patient's embolic load, the use of alpha-stat pH management for hypothermic bypass, efforts to minimise haemodilution by a reduction in circuit prime volume, the avoidance of rapid cerebral rewarming, and the prevention of perioperative hyperglycaemia. The rationale for the use of off-pump cardiac surgery to reduce the embolic load associated with the bypass circuit and therefore postoperative cognitive dysfunction has not been borne out with convincing evidence.

Are any other organ systems impacted?

Renal failure may be the result of inadequate oxygen delivery, depressed tubular function, intravascular haemolysis or microemboli. Its development is related to patient age, the degree of pre-existing kidney dysfunction and postoperative haemodynamic status. The requirement for dialysis postoperatively increases mortality risk markedly to approximately 60%. Haemodilution and diuresis secondary to hypothermia and mannitol-containing cardioplegia solutions frequently results in electrolyte derangement.

Visceral and splanchnic blood flow is compromised early in the setting of systemic hypoperfusion, and may be worsened by the use of high doses of vasopressors. Hepatic injury, mesenteric ischaemia or perforation and GI bleeding may all be the consequence of prolonged bypass times, inadequate tissue perfusion and raised venous pressures.

What is your approach to the haemodynamically unstable patient who has returned from cardiopulmonary bypass?

To correctly identify and rapidly treat the many possible causes of haemodynamic instability in the post-op cardiac patient, a structured approach is required alongside excellent communication with all team members including the cardiothoracic surgeon, cardiac anaesthetist and intensive care unit nurses. It can be useful to proceed with an ABC framework, and by identifying the aetiology of shock. Postoperative causes of hypovolaemic, distributive, cardiogenic, obstructive and neurogenic shock are given in Table 2.1.4.1. A high index of suspicion for bleeding and cardiac tamponade is required.

When assessing the airway, I would assess for patency and adequacy of ventilation, and identify possible threats to these. If the patient has already been extubated, it is important to consider the need to re-intubate the trachea if a return to the operating theatre is anticipated.

Oxygenation and ventilation should be optimised to allow adequate oxygen delivery and attenuate any rise in pulmonary vascular resistance caused by acidosis, hypoxia or hypercarbia. Setting the optimal value for positive end expiratory pressure (PEEP) is important. Higher pressures reduce left ventricular afterload but decrease right ventricular preload and can increase pulmonary vascular resistance. Lower pressures may be

Table 2.1.4.1 Causes of shock in the post-cardiac patient

Aetiology of shock	Cause
Hypovolaemia	Bleeding Inadequate volume resuscitation Diuresis
Distributive	Systemic inflammatory response Excessive sedation Vasodilators (GTN, sodium nitroprusside)
Cardiogenic	Bradycardia (hypothermia) Bradyarrhythmia (AV block) Tachyarrhythmia (AF, SVT, VT) Myocardial ischaemia Myocardial stunning Right ventricular failure Acute valvular failure Excessive left ventricular afterload Left ventricular outflow tract obstruction Hypocalcaemia
Obstructive	Cardiac Tamponade Tension Pneumothorax Massive Pulmonary Embolism
Neurogenic	Spinal cord infarction

insufficient to prevent alveolar collapse and therefore hypoxia and hypercarbia. It is important to rule out tension pneumothorax as an obstructive cause of shock. Extubated hypoxic patients who are intolerant of non-invasive ventilation may require reintubation.

To assess the cardiovascular status of the patient it can be useful to consider their preload, heart rate and rhythm, contractility, and afterload.

Inadequate preload may be the result of insufficient volume resuscitation, ongoing haemorrhage, cardiac tamponade, ventricular diastolic dysfunction, mitral or tricuspid valve failure, or vasoplegia. Diastolic dysfunction is common following cardiopulmonary bypass and therefore higher ventricular filling pressures may be required with further intravenous fluids. Milrinone and levosimendan can be used to improve lusitropy. Significant haemorrhage should be ruled out by monitoring drain outputs and obtaining serial haemoglobin measurements. Bedside tests of haemostasis like thromboelastography can be used to identify and treat coagulation abnormalities that require either protamine, clotting factors, fibrinogen or platelets to correct. Consideration should be given to early transoesophageal echocardiography in any haemodynamically unstable patient following cardiac surgery to diagnose tamponade or valve failure. Judicious use of vasopressors will improve diastolic perfusion and correct vasoplegia without excessively increasing left or right ventricular afterload. Excessive preload can also be problematic, particularly for the right ventricle, and so diuresis may be required.

Bradycardia may contribute to insufficient cardiac output to meet the patient's demand, and is common following hypothermia. Conduction system oedema may also cause atrioventricular block. Both can be rectified with pacing, and this is commonly performed via epicardial pacing wires, aiming for a heart rate of around 90 beats/min. Atrial pacing modes are preferred as they preserve the atrial contribution to ventricular filling. If these are not available, transvenous or transcutaneous pacing can be considered, alongside positive chronotropes such as dobutamine. In the setting of diastolic dysfunction, tachycardia is tolerated poorly as it reduces diastolic ventricular filling time, especially if there is concurrent loss of atrial conduction. Prophylactic electrolyte replacement is therefore undertaken, aiming to replace magnesium and potassium levels to above 1 mmol/L and 4 mmol/L respectively. If atrial or ventricular tachyarrhythmia occurs and the patient is haemodynamically compromised, they should receive synchronised DC cardioversion. Pharmacological options most commonly include the use of beta blockers or amiodarone to either rate control or restore sinus rhythm.

It is common for impaired myocardial contractility to be caused by myocardial stunning, with the right ventricle particularly susceptible due to its sensitivity to acute changes in preload or afterload. However, before starting inotropic agents it is important to exclude myocardial ischaemia due to graft failure, which may be identified on a postoperative ECG or with new regional wall motion abnormalities on echocardiography. This may require percutaneous coronary intervention, an intra-aortic balloon pump or a return to theatre. Calcium is an effective inotrope, and hypocalcaemia should be corrected.

Excessive ventricular afterload may cause low cardiac output state, with different factors affecting each ventricle. Right ventricular failure commonly causes low cardiac output state, and raised pulmonary vascular resistance may be contributory, with causes including hypoxia, hypercarbia, acidosis, hypothermia, left atrial hypertension or excessive vasopressor use. Pulmonary vasodilators like milrinone and inhaled prostacyclin or nitric oxide may all reduce right ventricular work, and right ventricular preload should be optimised with either fluids or diuretics. Elevated systemic vascular resistance increases left ventricular work, with causes including pain, hypothermia and excessive vasopressor use. Analgesia, rewarming, and the use of vasodilators or inodilators may all reduce left ventricular afterload. An important additional cause affecting the left ventricle is left ventricular outflow tract obstruction, in which the ejection of blood from the left ventricle is impaired by the dynamic closure of the outflow tract during systole. This can occur when an underfilled hypertrophied ventricle is subjected to excessive inotropy, or when there is systolic anterior motion of the mitral valve. It is an important cause to consider if the patient deteriorates after an inotrope is given. Treatment involves withdrawing the inotrope, fluid resuscitation and increasing the systemic vascular resistance with pure alpha-agonists like phenylephrine which help to splint open the outflow tract and increase diastolic filling time. Finally, inadequate afterload to allow forward perfusion can result from aortic or pulmonary valve failure, which can be diagnosed on echocardiography.

Optimal patient sedation and analgesia will prevent reductions in preload or excessive afterload respectively. In the setting of inadequate cardiac output to match the body's oxygen demand, consideration should be given to deep sedation and paralysis to reduce whole body oxygen consumption.

How would you diagnose and manage cardiac tamponade?

A high index of suspicion for cardiac tamponade is required in any post-op cardiac surgery patient with signs of a low cardiac output state. This may be indicated by hypotension requiring escalating vasopressor doses, reduced urine output, skin mottling, altered conscious level, rising lactate or worsening metabolic acidosis.

Cardiac tamponade can be difficult to diagnose. Given enough time, the pericardium can accumulate significant volumes of fluid before having any haemodynamic impact, for example in the setting of malignant pericardial effusions. However, if rapidly accumulated, relatively small volumes of pericardial fluid, blood or clot may significantly impair cardiac function.

Beck's triad comprises elevated central venous pressure, muffled heart sounds and shock, but this has low sensitivity. Breathlessness is the most sensitive symptom but is not specific. Orthopnoea and tachycardia are common. In cardiothoracic ICU, a rising central venous pressure in combination with evidence of shock may be suggestive. Classical clinical signs of tamponade include pulsus paradoxus, defined as a drop in systolic blood pressure of more than 10 mmHg during inspiration due to exaggerated ventricular interdependence, muffled heart sounds, small volume complexes on ECG and distended neck veins, which may not be present if there is concurrent hypovolaemia.

Echocardiography forms the mainstay of diagnosis, but following surgery the trans-thoracic approach is often impaired by the presence of surgical drains, pacemaker wires and residual air in the thorax. The transoesophageal approach is therefore more sensitive, and diagnostic features include the presence of a pericardial effusion (which may be localised), dilated inferior vena cava with loss of respiratory variation, increased respiratory variation of intracardiac blood flow and collapse of the cardiac chambers. Chamber collapse occurs at the point of lowest pressure during the cardiac cycle, and so right atrial systolic collapse and right ventricular diastolic collapse occurs first. Increasing intrapericardial pressure eventually compresses all of the cardiac chambers. It is important to realise that cardiac tamponade remains a clinical diagnosis, and cannot be conclusively ruled in or out with echocardiography alone.

Once the diagnosis is confirmed, definitive treatment relies on evacuating the pericardial blood or clot. This can be done with pericardiocentesis, but in the post-cardiac surgery patient it often entails re-sternotomy. Excellent communication and teamwork are required by the intensive care staff, cardiothoracic surgeon, cardiac anaesthetist, perfusionist and theatre team in order to expedite a return to theatre. The use of a cardiothoracic emergency page or cardiac arrest call may be useful.

In the absence of cardiac arrest there will be time to optimise the patient's haemodynamics before returning to theatre. They should receive 100% oxygen, have secure large bore IV access and cross-matched blood available. Emergency re-sternotomy equipment should be prepared and available at the patient's bedside in case of deterioration, or if they are too unstable to move to theatre. Right ventricular function may be improved with judicious use of IV fluids to optimise preload, and the reduction of afterload via the avoidance of high airway pressures, hypoxia and hypercarbia. Hypocalcaemia should be rectified to improve contractility. The use of inotropes must be balanced against the risk of arrhythmia which will be poorly tolerated. The extubated patient will require careful induction of anaesthesia with reduced doses of a cardiostable induction agent like ketamine or etomidate. Given the potential for haemodynamic

instability this should be done in theatre with the cardiothoracic team scrubbed and ready to operate.

Are there any differences in the management of cardiac arrest in the post-cardiac surgery patient?

Cardiac arrest is managed differently in post-cardiac surgery patients, reflecting the difference in their underlying cause. Common reversible precipitants include ventricular fibrillation, cardiac tamponade and bleeding. The Cardiac Advanced Life Support (CALS) algorithm is shown in Figure 2.1.4.1. A delay of up to one minute in starting external chest compressions is advocated in favour of three stacked DC shock attempts or external pacing, depending on the underlying rhythm. Emergency re-sternotomy should be performed within 5 minutes of arrest to facilitate evacuation of pericardial clot or internal cardiac massage. Lastly, conventional cardiac arrest doses of adrenaline (i.e., 1 mg IV) should not be used due to concern for rebound hypertension, bleeding or anastomotic disruption following return of spontaneous circulation. These modifications reflect the greater incidence of dysrhythmia as a cause of arrest, as well as the potential for myocardial injury or graft disruption from external chest compressions. To expedite timely re-sternotomy, the CALS algorithm allocates six key roles to staff members in a cardiac arrest scenario in the cardiothoracic intensive care unit (see Figure 2.1.4.2).

What are the goals of inotrope and vasopressor use following cardiac surgery?

Vasopressors are used following cardiac surgery to ameliorate some of the haemodynamic effects of the systemic inflammatory response. Specifically, they can improve vasoplegia and preload via constriction of arteries and veins respectively, thereby increasing coronary artery perfusion pressure and improving ventricular diastolic filling. The ideal vasopressor will not significantly increase ventricular afterload.

Inotropes can be used to maintain cardiac output in the setting of postoperative diastolic dysfunction and myocardial stunning. The goals of therapy are to improve cardiac output via positive chronotropy and increased myocardial contractility while maintaining sinus rhythm and without significantly increasing oxygen consumption.

Commonly used agents include noradrenaline, vasopressin, dobutamine, adrenaline, milrinone and levosimendan (Table 2.1.4.2). Noradrenaline is often the first-line vasopressor agent, but the VANCS trial, conducted in shocked post-cardiac surgery patients, suggested that vasopressin may be associated with fewer complications, specifically atrial fibrillation and acute kidney injury. Vasopressin may also be preferential in the setting of vasoplegia with pulmonary hypertension, as it does not increase pulmonary vascular resistance.

Dobutamine mainly increases cardiac output via positive chronotropy. It is generally the first-line inotropic agent, but has not been shown to be superior to milrinone in the setting of cardiogenic shock. Milrinone and other inodilators have the beneficial effects of reducing left and right ventricular afterload alongside a reduced tendency to arrhythmia versus traditional beta agonists. Levosimendan is an inodilator with the useful property of improving contractility without increasing myocardial oxygen consumption.



CARDIAC ARREST

assess rhythm

ventricular
fibrillation or
tachycardia

DC shock
(3 attempts)

asystole or
severe
bradycardia

pace
(if wires
available)

pulseless
electrical
activity

start basic life support

amiodarone
300mg
via central
venous line

consider
external
pacing

if paced, turn
off pacing to
exclude
underlying VF

prepare for emergency resternotomy

continue CPR with
single DC shock
every 2 minutes until
resternotomy

continue CPR
until
resternotomy

continue CPR
until
resternotomy

airway and ventilation

- If ventilated turn FiO₂ to 100% and switch off PEEP.
- Change to bag/valve with 100% O₂, verify ET tube position and cuff inflation and listen for breath sounds bilaterally to exclude a pneumothorax or haemothorax.
- If tension pneumothorax suspected, immediately place large bore cannula in the 2nd rib space anterior mid-clavicular line.

DO NOT GIVE EPINEPHRINE unless a senior doctor advises this.

If an IABP is in place change to pressure trigger.

Do not delay basic life support for defibrillation or pacing for more than one minute.

Figure 2.1.4.1 The Cardiac Advanced Life Support (CALS) algorithm.

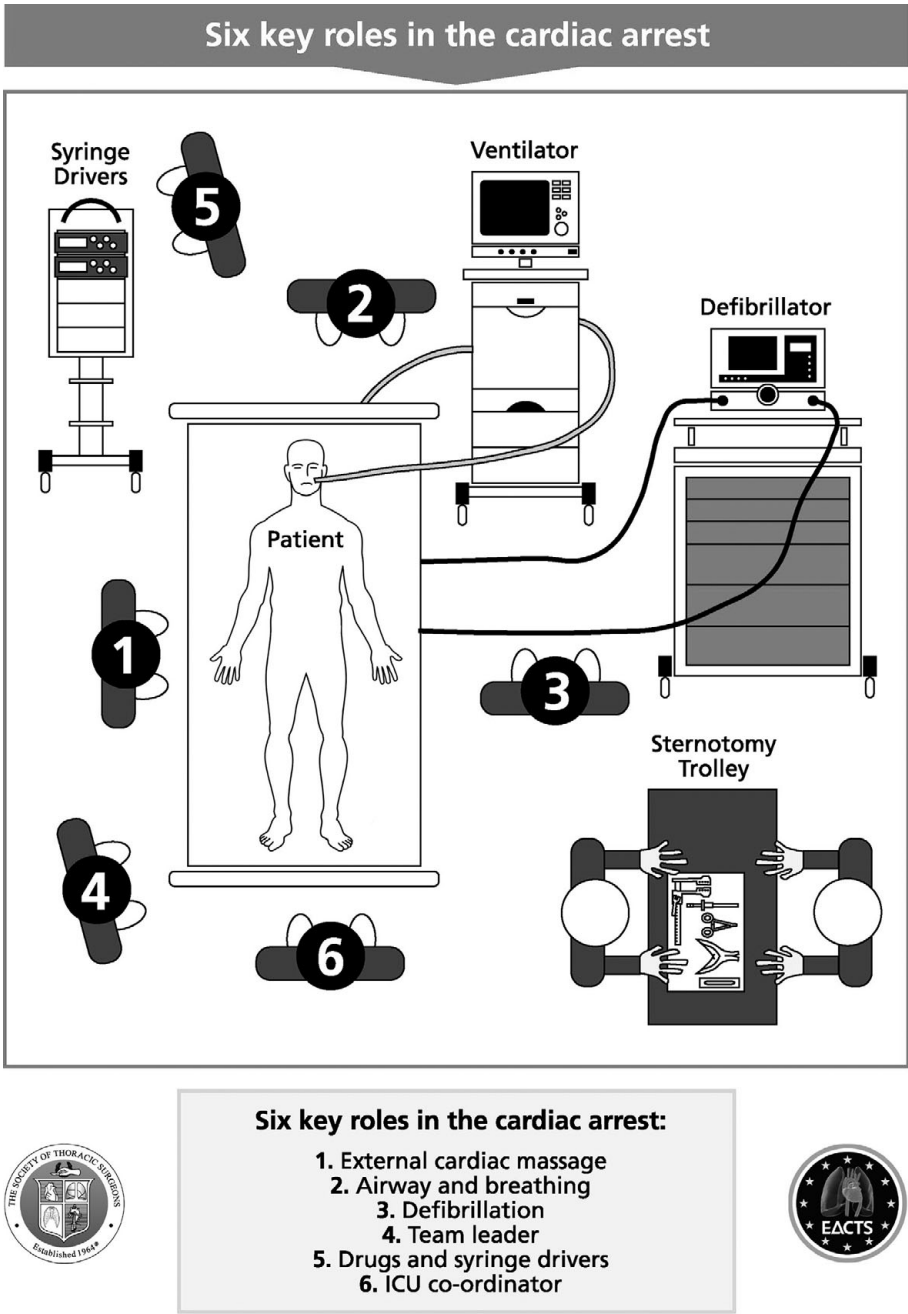


Figure 2.1.4.2 Six key roles of staff members on a cardiothoracic intensive care unit in the event of a cardiac arrest scenario.

Table 2.1.4.2 Properties of commonly used inotropes and vasopressors

	Noradrenaline	Vasopressin	Dobutamine	Milrinone	Levosimendan
Class	Endogenous Catecholamine	Posterior Pituitary neuropeptide	Synthetic catecholamine	Bipyridine inodilator	Pyridazinone-dinitrile derivative
Mechanism of Action	Alpha-1 receptor agonist. Beta-receptor effects at high doses	Agonist at V1 receptor (vasoconstriction) and V2 receptors (ADH effect)	Beta-1 and Beta-2 receptor agonist Partial alpha-1 receptor agonist	PDEIII inhibitor	Increases myocardial calcium sensitivity by binding troponin C in an energy neutral process Activates ATP-sensitive K ⁺ channels, causing vasodilation
Contractility	↑ (higher doses)	No effect	↑	↑	↑
Chronotropy	Reflex ↓HR (low dose)	Reflex ↓HR	↑	↑	↑
Effect on Cardiac Output	↑	↓ at high doses	↑	↑	↑
Lusitropy	No effect	No effect	↑	↑	↑
Effect on SVR	↑ linearly	↑ (non-linear). No increase beyond 0.04 units/min	SVR↓ at high doses but MAP maintained	↓	↓
Effect on PVR	↑	No effect	No effect	↓	↓
Myocardial O₂ consumption	↑	↑	↑	No effect due to reduced afterload	No effect
Pharmacokinetics	Short-acting and rapid onset. Rapid metabolism by COMT and MAO	Rapid onset. Majority renally excreted with half-life of approx. 20–30 mins	Short-acting and rapid onset. Rapid metabolism by COMT and MAO	Renally cleared with prolonged half-life	Hepatic metabolism to a potent metabolite with a long half-life (80 hours)
Other	Reduced catecholamine sensitivity following bypass and in acidosis	Potentiates effect of noradrenaline Receptor sensitivity preserved in acidosis	Reduced catecholamine sensitivity following bypass Increased CO largely via increase in HR	No change in sensitivity following bypass	Often given with an initial loading dose Effect takes 2–3 days to develop and lasts 2–3 weeks

Its effects take approximately three days to develop and, through its breakdown to a potent metabolite with a long half-life, can be effective for two to three weeks. It is often used as a pre-treatment in patients with low ejection fraction prior to cardiac theatre, in whom it may reduce the likelihood of developing a postoperative low cardiac output state.

Further Reading

A Practical Approach to Cardiac Anaesthesia, 4th ed. Hensley, Martin and Gravlee: Chapter 9 Postoperative care of the Cardiac Surgical Patient, and Chapter 19 Pathophysiology of Cardiopulmonary Bypass.

Brand J et al. Management of cardiac arrest following cardiac surgery. *BJA Education* 2018; 18(1): 16–22.

Hajjar LA et al, Vasopressin vs Noradrenaline in pts with Vasoplegic shock after cardiac surgery: The VANCS RCT. *Anaesthesiology*. 2017; 126: 85–93.

Jha A et al. Vasoactive therapy in shock. *BJA Education*. 2021; 21(7): 270–277.

Khan NE et al: A randomized comparison of off-pump and on-pump multivessel Coronary Artery Bypass Surgery. *New England Journal of Medicine*. 2004; 350: 21–28.

Madhivathanan PR et al. Perioperative implications of pericardial effusions and cardiac tamponade. *BJA Education*. 2020, 20(7): 226–234.

Mangano CM et al, Renal dysfunction after myocardial revascularization: Risk factors, adverse outcomes and hospital resource

utilization. *Annals of Internal Medicine*. 1998; 128: 194–203.

Mathew R et al. Milrinone as compared with Dobutamine in the treatment of cardiogenic shock. *New England Journal of Medicine*. 2021; 385: 516–525.

Mehta RH et al, Levosimendan in patients with left ventricular dysfunction undergoing cardiac surgery. *New England Journal of Medicine*. 2017; 376(21): 2032–2042.

Scarth E, Smith S. *Drugs in Anaesthesia and Intensive Care* 5th Edition 2016; Oxford University Press.

Shroyer AL et al. For the veterans affairs randomised on/off bypass (ROOBY) study group. On pump vs off-pump CABG surgery. *New England Journal of Medicine*. 2009; 361: 1827–1837.

Singh S, Hutton P, Cerebral effects of cardiopulmonary bypass in adults. *BJA Education* 2003; 3(4):115–119.

Tayama E et al. Arginine Vasopressin is an ideal drug after cardiac surgery for the management of low systemic vascular resistant hypotension concomitant with pulmonary hypertension. *Interactive Cardiovascular and Thoracic Surgery*. 2007; 6(6): 715–719.

2.1.5 Anaesthesia for Bronchoscopy – Neil Rasburn and Zoe Riddell

You have a 76-year-old man listed for rigid bronchoscopy. What information is important in the preoperative assessment?

Patients listed for bronchoscopy fall into two categories:

1. Those having an isolated diagnostic procedure for screening or sampling such as lung cancer staging or biopsy.
2. Those having bronchoscopy for a therapeutic procedure such as tracheal dilatation, stent insertion or foreign body removal.

It is essential to evaluate the upper airway and assess for symptoms and signs of obstruction, such as stridor and breathlessness at rest. A cardiovascular and respiratory history is also very important, as patients with lung cancer are often elderly smokers.

What investigations would you request?

Investigations I request will be both patient- and procedure-specific. These include:

- Chest X-ray: this is essential to assess lung pathology and see if the trachea is deviated.
- CT and MRI scans: may be indicated if there is evidence of airway obstruction or narrowing; however, these must be recent as airway pathology can change rapidly.
- Lung function tests are helpful if significant obstruction. The flow-volume loop can be particularly useful.
- ECG: essential if history of smoking and other cardiovascular risk factors.
- Routine bloods
- Lateral neck X-rays if there is a history of neck problems or rheumatoid arthritis, as rigid bronchoscopy requires significant neck extension.

Does bronchoscopy always require a general anaesthetic?

No. Flexible bronchoscopy can be performed under sedation and topical anaesthesia.

What anaesthetic technique would you use for this patient?

There is no one correct answer, think about what conditions you are aiming to achieve and what equipment you have available. Structure your answer into induction, maintenance and postoperative management.

The aims of anaesthesia for bronchoscopy are to provide anaesthesia with abolished airway reflexes, muscular relaxation, safe ventilation, and rapid emergence.

I would ensure standard AAGBI monitoring is in place. If the patient had significant cardiovascular disease, I would insert an arterial cannula prior to induction. I would attach depth of anaesthesia monitoring, such as entropy.

I would use IV glycopyrrolate as an anti-sialagogue premedication, and then anaesthetise and maintain anaesthesia using total intravenous anaesthesia with propofol and remifentanyl. This will have the advantage of providing good anaesthesia and obviating the profound vasopressor response associated with bronchoscopy.

I would use rocuronium as a muscle relaxant as this will provide deep relaxation throughout the procedure, but also enable me to fully reverse the patient with sugammadex at the end.

I would spray the cords with 4% lidocaine to prevent postoperative laryngospasm.

The patient should be positioned in the supine position with their head on a single pillow. I would ensure the eyes are protected and monitor the degree of neck extension required as the bronchoscope is advanced.

What ventilator strategies can you use?

The most important thing to recognise is that the airway is shared in this procedure. There are two possible ventilation strategies that can be employed, intermittent positive pressure ventilation (IPPV) via the ventilating arm of the bronchoscope or jet ventilation. Jet ventilation can be manual or high frequency. Manual jet ventilation is more common for bronchoscopy due to the short duration of the procedure.

Can you go into more detail about these differing techniques?

The Manujet or Sanders' injectors are used to provide manual jet ventilation. They use a high-pressure oxygen supply to release a jet of oxygen from a needle at the operator end of the bronchoscope. This creates a venturi effect, entraining atmospheric air and producing a positive pressure at the distal end of the bronchoscope. Intermittent inflation/deflation at a rate of approximately 8–10 breaths/min provides satisfactory oxygenation and carbon dioxide clearance. Adequacy of ventilation is based on visual chest rise, and it is essential that there is free passage of air from the upper airway to allow for both entrainment of air and passive expiration, thereby avoiding volutrauma. The needle size is important, as if too large, it can cause dangerous barotrauma.

Automated jet ventilation is via a ventilator that generates respiratory rates of 60–300 breaths/min. The respiratory rate, inspiratory time and driving pressure can be varied to ensure adequate oxygenation. Much lower tidal volumes and pressures are generated which can be advantageous in conditions such as bronchopleural fistula.

The ventilating bronchoscope has a side arm that can be connected to an anaesthetic circuit via a swivel connector, allowing IPPV. This technique is more common in infants and children, using a T-piece and hand ventilation, as there is minimal risk of barotrauma.

How would you maintain anaesthesia?

While volatile agents can be used with a ventilating bronchoscope, there is often a significant leak making it difficult to monitor end-tidal concentrations with a potential increased risk of awareness. There is also significant pollution of the theatre environment. TIVA is therefore a better option not only for IPPV but a necessity when using jet ventilation.

What are the postoperative considerations?

Unless bronchoscopy is followed by a procedure, muscle relaxation must be fully reversed, and a rapid emergence is desirable. The patient should be nursed with an oxygen facemask in the lateral position, with the diseased side down.

Potential complications include dental damage, aspiration of blood, awareness, pharyngeal rupture, airway rupture, cardiac arrhythmias, hypertension and myocardial ischaemia.

Be prepared to expand on the complications.

How do patients with inhaled foreign bodies present?

This problem frequently occurs in children under the age of five, but can present at any age, particularly in the obtunded. The presentation is variable depending upon the type, size and location of the foreign body, and the time since inhalation. It can be acute,

presenting with airway obstruction, or of a more insidious onset, presenting with a chronic cough, recurrent chest infection that fails to improve following antibiotic therapy, and unilateral wheeze.

Is a chest X-ray indicated in this clinical scenario?

If the patient presents with acute airway obstruction, there may not be time to perform a CXR, but this is unusual. The CXR could be normal or may show a radio-opaque object. In chronic cases, there may be nonspecific changes of atelectasis and consolidation. Inspiratory and expiratory films may reveal hyperinflation with the foreign body acting as a ball-valve in the main bronchus.

How would you anaesthetise a patient with an inhaled foreign body?

Induction options are either inhalational or intravenous. The traditional approach is an inhalational induction as this prevents positive pressure ventilation pushing the foreign body more distally. However, with the removal of halothane, the depth of anaesthesia required may make the patient apnoeic. Intravenous induction with fentanyl 0.5 mcg/kg followed by propofol 1mg/kg initial bolus can also be used until the patient is unconscious. Dexmedetomidine 1 mcg/kg can also be used as an induction agent.

Maintenance of anaesthesia can be with volatile agents using the sidearm of the ventilating bronchoscope connected to the circuit; however, this can pollute the theatre environment. I would therefore maintain anaesthesia using IV agents using TIVA with remifentanyl and propofol, aiming to still maintain spontaneous ventilation. I would also administer dexamethasone IV to reduce airway swelling and spray the vocal cords with lidocaine using a mucosal atomisation device.

Oxygenation of the patient can be through the side arm of the bronchoscope or with the advent of high-flow nasal cannula, Transnasal Humidified Rapid – Insufflation Ventilatory Exchange (THRIVE) can be used.

What are the postoperative complications?

The main concern is laryngeal stridor. This can be managed with nebulised adrenaline, 5 ml of 1:10,000, repeated at 2–4 hours and IV dexamethasone 600 mcg/kg/day in four divided doses. Severe stridor may necessitate reintubation. If this is required, a smaller endotracheal tube should be used.

What other bronchoscopic interventions are you aware of?

In critical airway stenosis, tracheo-bronchial stents can be inserted either as a palliative procedure or until definitive surgery can take place.

Resection of tumours using mechanical, diathermy, laser or cryotherapy are also common procedures. Lastly treatment of massive haemoptysis can be via rigid bronchoscopy too.

What are the important anaesthetic considerations for these procedures?

Preoxygenation is essential in these patients, as airway obstruction on induction of anaesthesia is possible. Intravenous or inhalational induction both have advantages and disadvantages. The most important aspect is to have a skilled bronchoscopist on hand, as this may be the only way to relieve the obstruction.

If using a laser, routine precautions should be used including:

- Using the lowest concentration of inspired oxygen
- Avoiding nitrous oxide
- Using short bursts of laser treatment
- Using a metal bronchoscope avoids the need for a metal or foil wrapped tube.

You mentioned earlier that flexible bronchoscopy is performed under local anaesthetic and sedation, do you ever come across this technique in your practice?

Yes, this technique is performed for awake fibreoptic intubation.

What are the indications for awake fibreoptic intubation?

Known or anticipated difficult airway

- Unstable Cervical spine injury or unstable neck, for example in rheumatoid arthritis with a risk of aspiration
- Obesity and obstructive sleep apnoea can also be considered
- Progressive head and neck pathology with impending airway compromise.

Are there any contraindications?

Yes

- Patient refusal
- Local anaesthetic allergy
- Airway bleeding
- Relative contraindications of using the nasal route include coagulopathy and base of skull fracture
- Periglottic masses: there is a risk of complete airway obstruction or laryngospasm.

Is the nasal route best?

This route is preferred, in my experience as it is frequently easier in the group of patients with limited mouth opening, and the oral route gives a poor angle of approach to the larynx. However the Difficult Airway Society guidelines for awake tracheal intubation also advocate for awake videolaryngoscopy via the oral route.

And how would you anaesthetise the airway for this procedure?

There are several ways to perform this procedure. It can be done with or without sedation. The technique with which I am most familiar is according to the Difficult Airway Society Awake Tracheal Intubation Guideline.

1. I would perform this procedure in an operating theatre with an ENT surgeon in the theatre.
2. I would position the patient at 45 degrees, apply full AAGBI monitoring and give oxygen via a nasal catheter.
3. I would start to topicalise the airway, noting the maximum dose of lidocaine for the patient. I would topicalise the nostrils using co-phenylcaine spray – up to 2 ml.

4. I would then use 10% lidocaine to topicalise the oropharynx, tonsillar pillars and base of tongue, spraying 20–30 times over 5 minutes, during inspiration.
5. After non-traumatically testing the topicalisation, I would sedate the patient using a remifentanyl TCI, starting at an effect site concentration of 1 ng/ml and titrating up to 3 ng/ml.

How would you anaesthetise the vocal cords?

I would use 2 ml of 2% lidocaine, and spray above, at and below the cords via an epidural catheter threaded through the working channel of the bronchoscope.

Are you aware of any specific nerve blocks that can be performed?

Superior laryngeal nerve blocks can be performed either by injecting local anaesthetic bilaterally, just above the thyroid cartilage at a point one-third of the distance between the midline and the superior cornu.

Alternatively, pledgets soaked in local anaesthetic can be placed into each piriform fossa using forceps. I have never witnessed the use of these blocks in my clinical practice.

Further Reading

Ahmad I, El-Boghdadly K, Bhargava R, Hodzovic I, McNarry AF, Mir F et al. Difficult Airway Society Guidelines for awake tracheal intubation (ATI) in adults. *Anaesthesia*. 2020;75(4):509–528.

Bould MD. Essential Notes: The anaesthetic management of an inhaled foreign body in a child. *BJA Education*. 2019;19(3):66–67.

Chadha M, Kulshrestha M, Biyani A. Anaesthesia for bronchoscopy. *Indian Journal of Anaesthesia*. 2015;59(9):565–573.

2.1.6 One-Lung Ventilation – Carl J Morris and Zoe Riddell

What are the indications for one-lung anaesthesia?

Indications for one-lung anaesthesia can be classified into absolute and relative.

The absolute indications can be further classified:

1. Prevention of damage or contamination of the healthy lung from pathology in the contralateral lung such as:
 - Infection or lung abscess
 - Pulmonary haemorrhage
 - Other fluid
2. Controlling distribution of ventilation in the case of:
 - Large bronchopleural fistula
 - Risk of rupture of large bulla
 - Traumatic bronchial injury
3. Facilitating single-lung ventilation for procedures such as:
 - Single-lung lavage
 - Video-assisted thoracoscopy (VATS)
 - Surgery to proximal bronchial tree

The relative indications are to improve surgical access, and these can be classified into strong and weaker indications.

Strong Indications include:

- Thoracic aortic aneurysm
- Pneumonectomy
- Lung volume reduction surgery
- Minimally invasive cardiac surgery.

Weaker indications include:

- Oesophageal surgery
- Middle and lower lobectomy
- Mediastinal mass reduction.

How would you achieve one-lung ventilation?

The most common method I am familiar with is the use of a double lumen endobronchial tube. There are also techniques where a single lumen endotracheal tube combined with a bronchial blocker can achieve lung isolation. In an emergency or in a difficult airway scenario, a deliberate endobronchial intubation with a long single lumen tube technique has also been used.

Can you think of advantages and disadvantages to the use of bronchial blockers?

Hopefully this type of question means the SOE is going well and they are exploring your knowledge a little further.

Bronchial blockers are useful when there is difficult airway anatomy meaning it is extremely difficult or impossible to pass a double lumen tube. They can be used for lobar isolation. In patients already intubated with a single lumen endotracheal tube or tracheostomy, a bronchial blocker avoids the need for re-intubation. They allow easy post-operative dual lung ventilation as you can simply withdraw the blocker.

The disadvantages are that they take more time to accurately position to allow one-lung ventilation, they can frequently become dislodged intraoperatively, they do not give easy access to the deflated lung (for example for suctioning or bronchoscopy), lung collapse of the non-dependent lung is often slow or incomplete and they can be difficult for left-sided one-lung ventilation.

Would you have a preference between a left or a right-sided double lumen tube?

I would prefer to insert a left-sided tube. This is because the adult distance from the carina to the left upper lobe bronchus is about 5 cm versus 2.5 cm on the right. Therefore, a left-sided tube is much less likely to cause upper lobe obstruction.

What are the indications for a right-sided double lumen tube?

Indications include surgery that involves the left main bronchus including left pneumonectomy, lung transplant, left tracheobronchial disruption and left-sided

thoroscopic surgery. Other indications are patients with distorted anatomy of the left main bronchus such as tumour compression or those with descending thoracic aortic aneurysms.

What type of double lumen endobronchial tube would you choose?

I have used the Portex and Mallinkrodt Bronchocath type.

And the size?

Adult double lumen tubes commonly range from 32 to 41F (French gauge). Traditionally a 37F would be used for the average sized women and a 39F for an average sized man; however, the bronchocath type tubes tend to be longer so I would go down a size to reduce the risk of airway trauma.

How would you insert a double lumen endobronchial tube?

I would assess the patient, in particular reviewing the notes for any imaging for potential difficulties with intubation.

I would carry out my standard preoperative checks of drugs and equipment, in particular checking both the tracheal and bronchial cuffs on the double lumen tube.

I would lubricate the outside of the tube and insert the stylet into the endobronchial lumen, making sure the tip does not protrude from the distal end.

I would ensure that I have a working bronchoscope to check the position or assist with a difficult intubation.

After induction of anaesthesia and administration of neuromuscular blockade, I would perform direct laryngoscopy to visualise the glottis.

I would insert the tube with the endobronchial section facing anteriorly to pass through the cords, then withdraw the stylet, rotate the tube 90 degrees to the left (if passing a left-sided tube) and advance slowly until resistance is met.

I would inflate the tracheal cuff and attempt to ventilate the patient's lungs, checking for a satisfactory end-tidal carbon dioxide trace.

Do you know what depth to insert a double lumen tube?

This is based on the formula: $12 + (\text{patient height in cm}/10)$ measured at the teeth. For example, for 170 cm patient, I would insert the tube to 29 cm.

And how would you confirm correct position?

I would always use a bronchoscope to check the position, but you can also do this clinically.

Using a bronchoscope, I would first look down the tracheal lumen to make sure I could visualise the carina, and check that the endobronchial lumen is in the correct bronchus, with the blue cuff visible just below and not herniating above the carina.

I would then check the endobronchial lumen confirming that the upper lobe bronchus was patent. This is particularly important with right-sided tubes, where I would check the alignment of the Murphy's eye with the right upper lobe bronchus.

Why do you use a bronchoscope?

In various studies, critical malpositioning has occurred in around 25% of cases. In the 1998 NCEPOD report, malpositioning of the double lumen tube was associated with 30% of oesophagectomy deaths and up to 12% of double lumen tubes become displaced intraoperatively.

Could you talk me through the clinical checks?

This is a three-step check (See Figure 2.1.6)

1. Inflate the tracheal cuff and perform positive pressure ventilation. Ensure you have a capnography trace. Visualise equal chest rise and auscultate to confirm bilateral air entry.
2. Stop ventilation through the tracheal limb by either clamping or closing the swivel connector and disconnect this limb from the DLT. Inflate the bronchial cuff slowly with 1–3 ml of air and ventilate through the bronchial lumen. Auscultate and visualise the chest to confirm unilateral ventilation and ensure there is no audible air leak.
3. Resume bilateral air entry by releasing the tracheal lumen clamp or opening the swivel connector. Visualise and auscultate the chest to confirm bilateral air entry.

What are the physiological consequences of one-lung ventilation?

One-lung ventilation usually takes place in the lateral decubitus position. The lower, dependent lung is ventilated, while the upper lung collapses on opening the chest.

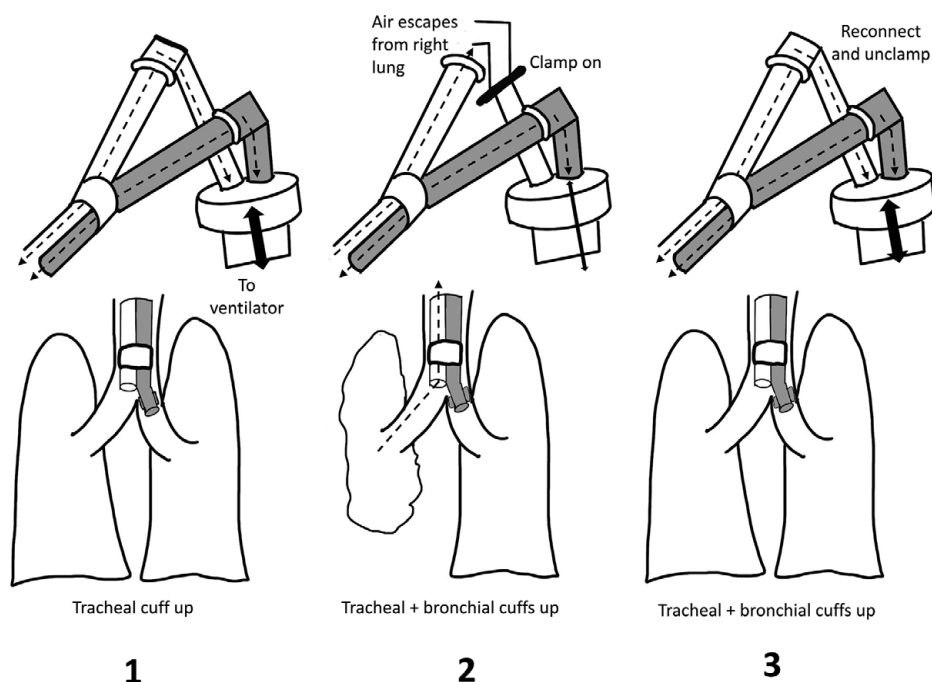


Figure 2.1.6 Clinical checks when inserting a double lumen tube.

Gravity aids perfusion of the dependent lung, but can also lead to compression of the airways, pooling of secretions and hypoxaemia.

Any blood flow to the non-ventilated lung, or areas of collapse in the ventilated lung lead to a shunt. The shunt fraction is estimated to be about 20–40%. Hypoxic vasoconstriction in the non-ventilated lung can also reduce the shunt fraction, but only by 50%. Dead-space volume, (i.e ventilated lung with no perfusion) can increase, especially if there is difficulty deflating the non-dependent lung, and this can contribute to hypercarbia.

Hypercarbia and a reduction of the arterial oxygen partial pressure result in a shift of the oxygen dissociation curve to the right (the Bohr Effect) leading to rapid dissociation of oxygen from haemoglobin.

How would you deflate the non-dependent lung?

I would clamp the catheter mount of the non-dependent lung and open the port to the same lung. I would then check with the surgeon or look on the screen, if a VATS procedure is being performed, to ensure the lung is deflating.

How would you adjust the ventilation when the non-dependent lung is deflated?

I would keep the tidal volumes the same as for two-lung ventilation (up to a maximum of 10 ml/kg), ensuring the peak airway pressures are below 30 cm of water. I would adjust the rate to ensure normocapnia. I would titrate the fraction of inspired oxygen down to a minimum of 0.5.

How would you deal with hypoxaemia?

I would first check there is no disconnection or blockage of the circuit.

I would then base my strategies on addressing the dependent and non-dependent lung separately.

Dependent lung strategies include:

- Increasing the fraction of inspired oxygen concentration (but this can also contribute to absorption atelectasis and may not improve oxygenation if there is a large shunt).
- Suctioning the ventilated lung to remove pooled secretions or blood.
- Re-checking the tube position, especially if the airway pressures are high, as the right upper lobe could be occluded.
- Cautious application of positive end expiratory pressure may improve oxygenation through recruiting collapsed alveoli; however, it can worsen any shunt by increasing blood flow to the non-ventilated lung.
- Ensuring adequate perfusion to the lung by treating hypotension with vasopressors or fluids.

Non-dependent lung strategies aim to decrease the shunt. These include:

- Insufflating oxygen to the non-ventilated lung using a suction catheter.
- Application of CPAP; however, this can cause expansion of the lung.
- Clamping the pulmonary artery; however, this may not be practical depending on the surgery.
- Failing these measures, returning to two-lung ventilation may be necessary.

What would you do if the patient became extremely hypoxic?

I would hand ventilate using 100% oxygen which would allow me to assess and treat simultaneously while calling for senior help. I would institute the measures described before, the last resort being to revert to two-lung ventilation.

Further Reading

Ahmed S, Janjua S, Ishaq M, Tariq M, Raza H. Double lumen intubation; Reliability of the auscultatory method? *Professional Medical Journal*. 2009; 16: 105–108.

Ashok V, Francis J. A practical approach to adult one-lung ventilation. *BJA Education*. 2018; 18(3): 69–74.

2.1.7 Anaesthetic Management of Patients with Transplanted Organs including Heart, Lungs and Kidneys – Asim Iqbal

This topic is well suited to the clinical viva, but the beginning may feature as part of a basic science viva.

Can you draw an overview of the sympathetic nervous system for me?

To avoid a long silence, describe to the examiner what you are drawing, while you are drawing it. Draw a picture of a brain in sagittal section with the spinal cord extending down. Parallel to the spinal cord, draw another line and label this the sympathetic chain. Label and draw that it starts at T1 and extends down to the L1/ L2 level. If you wish, you can draw the organs and glands supplied by the nerves in a column next to the sympathetic chain (starting from top to bottom: the eye, the parotid gland, the heart, the lungs, the stomach, the liver, adrenal glands and the bladder).

Where does the sympathetic chain of the heart arise from?

The origin of sympathetic chain of the heart arises from the T1 to T4 segments of the spinal cord.

What are these fibres called?

They are called the cardio-accelerator fibres.

What can you tell me about the physiology of the transplanted heart?

The Frank-Starling relationship between end-diastolic volume and cardiac output is normal and the transplanted heart is said to be preload dependent. Maintenance of a high or normal preload is desirable in the management of these patients. Coronary autoregulation also remains intact after heart transplantation.

What about the other autonomic or sensory influences?

The transplanted heart has no parasympathetic, sympathetic, or sensory innervation. All direct autonomic influences are absent. The loss of vagal tone produces a higher than normal resting heart rate, in the region of 90–110 beats/min. The Valsalva

manoeuvre and carotid sinus massage will not affect the heart rate. The baroreceptor reflex is also interrupted in the transplanted heart as the efferent limb (the vagus nerve) is denervated at the time of transplant. Other effects of cardiac denervation include the loss of the sympathetic response to laryngoscopy and endotracheal intubation. In terms of sensory changes, myocardial ischaemia and infarction may be silent due to the heart's denervated state. Patients usually undergo regular angiographic investigation for this complication. However, unpredictable re-innervation can occur after heart transplantation.

What about the response to circulating catecholamines?

Although the sympathetic fibres to the heart are also interrupted at the time of transplant, the response to circulating catecholamines remains normal or can even be enhanced secondary to denervation sensitivity. Catecholamine receptor density can be normal or increased. In a denervated heart, the catecholamine response is different from that in a normal heart because intact sympathetic nerves are necessary for the normal uptake and metabolism of catecholamines.

How does the transplanted heart respond to sympathomimetic drugs?

The transplanted heart responds well to directly acting sympathomimetics. Epinephrine and norepinephrine have an augmented inotropic effect in heart transplant recipients. Both drugs tend to have a higher β to α adrenoreceptor ratio. Isoprenaline and dobutamine have similar effects in both denervated and normal hearts, so both are effective inotropes in heart transplant recipients. They increase myocardial contractility more than dopamine, which acts predominantly by norepinephrine release. Dopamine is a less effective inotrope in the denervated heart, having predominantly α and dopaminergic effects. In terms of other drugs with an inotropic effect, digoxin will still increase contractility, but have minimal effect on the AV node.

What about indirectly acting drugs?

Directly acting agents are more effective than indirect ones, due to the lack of catecholamine stores in myocardial neurones. Indirectly acting drugs, such as ephedrine, have diminished responses on heart rate and blood pressure in heart transplant recipients. As vagolytic drugs will be ineffective in increasing heart rate, positively chronotropic drugs such as isoprenaline, should be available to hand.

Any other anaesthetic drugs interactions to be aware of?

Profound bradycardia and even cardiac arrest after neostigmine have been described, despite vagal nerve interruption. This is due to up regulation of the post-ganglionic nicotinic receptors from the loss of vagal innervation. Consideration should be given for the avoidance of neuromuscular block. If it is required, short-acting agents should be used. Newer reversal agents such as sugammadex should avoid the possible adverse responses to neostigmine. If anticholinesterase drugs are used, a muscarinic antagonist should always be co-administered (also to avoid the non-cardiac muscarinic effects) and potent β -adrenergic agonists such as isoprenaline or epinephrine should be readily available.

Describe the anaesthetic management of a heart transplant recipient?

Be ready to divide your answer into the preoperative, intraoperative and postoperative management of these patients.

The general considerations related to any transplant recipient should encompass the physiological and pharmacological problems of graft denervation, the potential for rejection, the adverse effects of immunosuppression, and to be cognisant of the risk or presence of infection. As up to 25% of these patients will have a permanent pacemaker, the usual considerations that apply to pacemakers may have to be taken into account. Along with taking a standard anaesthetic history, preoperative assessment should focus on evaluation of the functional status of the organ and detecting complications of immunosuppressive therapy.

The highest incidence of rejection occurs within the first three months after the transplant. Rejection may be apparent from the history, with a decreased exercise tolerance as an indication of deteriorating myocardial function, or from biopsy results. Mild rejection does not usually compromise cardiac contractility, but severe rejection may cause significant systolic and diastolic dysfunction. Other clinical indicators of rejection include fatigue, congestive cardiac failure, accelerated atherosclerosis, silent myocardial infarction on the ECG and ventricular dysrhythmias.

Accelerated atherosclerosis in the graft is a serious and common problem. This is a circumferential diffuse involvement of long coronary artery segments as opposed to focal plaques. It affects half of all heart transplant within three years and more than 80% at five years. As myocardial ischaemia or infarction are usually silent, the patient may have had a recent coronary angiogram. This may, however, underestimate the severity of atherosclerosis. Dobutamine stress echocardiography may conversely identify functional ischaemia without any significant lesions. It would be prudent to assume accelerated atherosclerosis in people who received a heart transplant more than two years ago.

A preoperative 12-lead ECG may demonstrate two p-waves – one representing the recipient's sinoatrial (SA) node which is usually left intact, and the other representing the donor's SA node. However, the more recent bicaval transplant technique has superseded this, resulting in one P-wave only. There may also be pacing spikes if a pacemaker is present.

What type of immunosuppressive therapy might the patient be receiving? Are there any particular problems associated with this?

Heart transplant recipients usually require more aggressive immunosuppressive regimens than recipients of other solid organ transplants. Immunosuppressive therapy may be a combination of prednisolone, mycophenolate, calcineurin inhibitors (such as cyclosporine, or tacrolimus) or mammalian target of rapamycin (mTOR) inhibitors (such as sirolimus) and azathioprine. Side effects of these drugs include steroid-related side effects, nephrotoxicity with renal impairment and subsequent hypertension, bone marrow suppression, hepatotoxicity and the development of opportunistic infections and malignancies. Consideration should be given for perioperative stress dose supplementation to patients on steroids.

At therapeutic levels, tacrolimus and cyclosporine can cause a dose-related decrease in renal blood flow and glomerular filtration rate, secondary to renal vasoconstriction.

Increases in thromboxane A2 and endothelin production are responsible for these altered renal haemodynamics.

Opportunistic infections secondary to immunosuppression may be bacterial, viral, fungal, or protozoan. However, reducing the dose of immunosuppressive drugs in the perioperative period may increase the risk of rejection. It is important to appreciate that the immunosuppressed patient may not present with the classical signs and symptoms of infection - leucocytosis, fever and other physical signs may be lacking.

Preoperative blood tests may reveal thrombocytopenia and leucopenia secondary to azathioprine. Tacrolimus or cyclosporine therapy can additionally cause hypomagnesaemia and hyperkalaemia.

Are you aware of any interactions between immunosuppressant therapy and anaesthetic agents?

Immunosuppressive drugs can interact with and alter the pharmacological behaviour of many anaesthetic drugs. Cyclosporine and tacrolimus are metabolised in the liver through the cytochrome P-450 system. Therefore, many drugs administered during anaesthesia or perioperatively can potentially affect cyclosporine or tacrolimus blood levels.

Cyclosporine enhances the effects of muscle relaxants and prolonged neuromuscular block has been described. Therefore, patients receiving this drug may require smaller doses of non-depolarising muscle relaxant. Patients on these drugs may also exhibit a greater response to standard doses of benzodiazepines.

How would you manage these patients intraoperatively?

There is no evidence to support one anaesthetic technique over another. Both regional and general anaesthesia have been successfully used for transplant recipients. However, the absence of reflex increases in heart rate can make the blood pressure particularly sensitive to rapid vasodilatation. If an epidural or spinal technique is to be used, the platelet count and clotting studies should be normal. Thrombocytopenia as a result of immunosuppressive therapy increases the risks associated with central neuraxial blockade.

What level of monitoring do you think is adequate for these types of patients?

The choice of perioperative monitoring techniques is determined by the type of surgery, the anaesthesia planned, and the equipment available. Central venous, intravenous and arterial access may be difficult due to previous repeated use. Invasive monitoring requires strict aseptic precautions and should be evaluated in terms of the risk-benefit ratio to the patient. If a central venous line is required, it would be desirable to avoid the right internal jugular vein, as this is often accessed for cardiac biopsies. There may be a role for perioperative transoesophageal echocardiography.

How would you manage the patient's fluid balance intraoperatively?

Hypovolaemia is poorly tolerated due to the preload dependence, so maintenance of a high or normal preload is important. However, this must be balanced against clinically

significant reductions of tacrolimus or cyclosporine serum levels, which can be caused by dilution with a large perioperative fluid infusion.

How would you manage this patient postoperatively?

The preoperative state of the patient and the nature of the surgery will define the most suitable postoperative environment for the patient, but I would have a lower threshold for admitting them to a higher dependency area.

Non-steroidal anti-inflammatory drugs should be avoided due to the risk of adverse drug interactions. They augment nephrotoxicity of cyclosporine, as both drugs affect the renal microcirculation. Other interactions can result in gastrointestinal haemorrhage and hepatic dysfunction.

Immunosuppressive therapy should be continued during the perioperative period and daily monitoring of cyclosporine or tacrolimus blood levels is recommended. The dose of immunosuppressive drugs should not be altered unless the route of administration needs to be changed from oral to IV. Supplemental 'stress-coverage' doses of steroids is controversial, but is a relatively low-risk treatment and used frequently.

Strong consideration should be given to thromboprophylaxis in transplant recipients, especially if other risk factors are present.

The viva will now go on to discuss the anaesthetic management of lung, liver and kidney transplant recipients. The general considerations related to any transplant recipient mentioned above (the physiological and pharmacological problems of allograft denervation, the potential for rejection, the side effects of immunosuppressant drugs, and the risk or presence of infection) still apply. These questions in this section will focus on the specific problems encountered with these patients.

The Anaesthetic Management of a Lung Transplant Recipient.

How does denervation affect the lung?

The denervation of the lung(s) seems to have a minimal effect on the pattern of breathing, but it removes afferent sensation below the level of the tracheal anastomosis. Patients with a tracheal anastomosis lose their cough reflex and are therefore more prone to silent aspiration and retention of secretions. Bronchial hyperreactivity causing bronchoconstriction is a common occurrence. The ventilatory response to carbon dioxide remains normal. Pulmonary denervation does not interfere with hypoxic pulmonary vasoconstriction.

How could rejection be detected preoperatively?

Symptoms of rejection may be very similar to those of an upper respiratory tract infection and include fatigue, shortness of breath and fever. It can be difficult to differentiate between the two. Blood tests may reveal leucopenia, while an arterial blood gas may reveal hypoxaemia and an increased alveolar to arterial oxygen gradient. Rejection can also be detected with pulmonary functions tests. The forced expiratory volume, vital capacity, and total lung capacity would be significantly reduced. Higher morbidity has been demonstrated in elective surgery patients presenting during a rejection episode.

Obliterative bronchiolitis is thought to be caused by chronic rejection, and it usually presents after the third month post-transplantation. Post lung transplant bronchiolitis obliterans affects up to 50% to 60% of patients who survive five years after lung transplantation. Chest radiography may reveal perihilar infiltrates, opacification of the graft or evidence of air trapping on expiration. In this situation, pulmonary function testing would reveal an obstructive defect. A worsening obstructive respiratory pattern in the late course of obliterative bronchiolitis may pose serious anaesthetic challenges if surgery is required.

As transplanted lungs may have ongoing rejection which can adversely affect pulmonary function, patients should have preoperative spirometry tests. A recent CT scan may be valuable in aiding anaesthetic management. If infection or graft rejection is evident, elective surgery should be postponed and appropriate investigations undertaken.

Do you have any concerns with ventilating these patients?

Single-lung transplant recipients may cause specific concern, especially if the native lung is emphysematous. The native lung may be highly compliant and the donor lung may have normal or reduced compliance, causing an imbalance. With institution of positive pressure ventilation, dynamic hyperinflation of the emphysematous lung with haemodynamic instability and problems with gas exchange may develop. A double lumen tube or independent lung ventilation techniques may be required, to reduce the airway pressure and minute ventilation in the native lung. Care should be taken when positioning tubes to avoid tracheal or bronchial anastomosis sites. A fiberoptic scope may be useful for this. Nasal intubations may pose risk of infection from the nasal flora.

General lung protective ventilation strategies should be applied to lung transplant recipients, with respect to tidal volumes and PEEP. Limiting peak inspiratory to 30 to 35 cm/H₂O and plateau pressures to 20 to 25 cm/H₂O may prevent barotrauma to bronchial anastomoses and potentially diseased alveoli. Postoperatively, optimal respiratory care should be instituted.

Would you have any preference for regional or general anaesthesia?

As lung transplant recipients lack a cough reflex below the tracheal anastomosis level, they may have difficulty clearing secretions. In view of this abolished cough reflex, the increased risk of chest infection, and the potential for bronchoconstriction, one can reason that a regional technique, if possible, may be preferable to one that requires insertion of an endotracheal intubation.

Describe the fluid management of lung transplant recipients?

The lymphatic drainage in the transplanted lung is usually disrupted. This can predispose to interstitial fluid accumulation, particularly early on in the post-transplantation period. A careful and limited crystalloid infusion should be used perioperatively. In combined heart-lung transplant recipients, however, fluid management can be particularly problematic. While the heart requires an adequate preload to maintain cardiac output, the lungs may have an increased propensity towards developing pulmonary oedema. Invasive hemodynamic monitoring is often useful in this subset of patients.

The Anaesthetic Management of a Liver Transplant Recipient.

Describe what happens to liver function following transplantation?

Following a successful liver transplant, the tests of synthetic function of the liver should be normal. An initial significant increase in all liver enzyme levels gradually decreases to normal over the first two postoperative weeks as allograft function becomes normal. The recovery of the liver's capacity to metabolise drugs occurs almost immediately after reperfusion of the graft.

Liver transplantation reverses the hyperdynamic state that is characteristic of patients with end-stage liver disease, and cardiac performance improves in the months after transplantation.

What relationships are you aware of between the lung and the liver?

How might they change after transplantation?

Pulmonary dysfunction in patients with end-stage liver disease can result from:

- 1) intrapulmonary shunting caused by intrapulmonary vascular dilatation,
- 2) ventilation/perfusion mismatch (from pleural effusions, ascites, and increased closing capacities),
- 3) diffusion abnormalities caused by interstitial pneumonitis and/or pulmonary hypertension, and
- 4) impaired hypoxic pulmonary vasoconstriction.

After successful liver transplantation, oxygenation does improve in most patients. The hypoxaemia caused by ventilation/perfusion mismatch often resolves during the first few postoperative months. Patients with pre-existing true shunts may require more time to achieve reversal of hypoxaemia, or it may not resolve at all.

How would you detect rejection in the transplanted liver?

The clinical signs of rejection include fever, malaise, hepatosplenomegaly, ascites, and right upper quadrant pain. The most reliable indicators are an elevated bilirubin, AST and ALT.

Are there any other physiological considerations that may influence your management in somebody with a transplanted liver?

The normal physiological mechanisms that protect hepatic blood flow are diminished after liver transplantation. The liver functions as an important blood reservoir in hypovolaemic states by means of a vasoconstrictive response, and this mechanism can be markedly impaired following liver transplantation. I would be happy to use the common inhalational anaesthetic agents in use today, as there is no evidence of increased risk of developing hepatitis after their administration. Renal dysfunction is common in liver transplant recipients, and this has important pharmacological implications.

If the patient is returning to theatre in the immediate postoperative period and a blood transfusion is required, I would use blood products judiciously as hepatic arterial

thrombosis has been retrospectively associated with over-transfusion of blood products and haemoconcentration. If this complication occurs, the mortality rate is high in this transplant population. These patients should have minimal blood viscosity (haematocrit approximately 28%) during the perioperative period.

The Anaesthetic Management of a Kidney Transplant Recipient.

Describe the anaesthetic management of a patient who has had a renal transplant?

Although recipients with an adequately functioning kidney graft may have creatinine levels within the normal range, it is important to recognise that the glomerular filtration rate and effective renal plasma flow are likely to be significantly reduced. Hence, the activity of drugs that rely upon renal excretion may be prolonged and should be avoided.

During preoperative assessment, it is important to recognise that there is an increase in the incidence and severity of cardiovascular disease in this population, especially due to the success of renal transplantation in elderly and diabetic patients. Hypertension is a frequent finding in this patient population and it is common for renal transplant recipients to be taking oral antihypertensive therapy for this. Both of these should be managed accordingly.

How would you detect rejection of the kidney graft?

Uraemia, proteinuria, and hypertension may indicate chronic rejection of the graft. Patients with renal graft dysfunction may have also been recommenced upon haemodialysis.

Describe your intraoperative management?

If the patient has undergone recent haemodialysis, they may be hypovolaemic and/or hypokalaemic. Hypovolaemia leads to cardiovascular instability, and hypokalaemia causes cardiac arrhythmias and increased sensitivity to muscle relaxants. As the variables of renal function are likely to be abnormal in kidney transplant recipients, it is sensible to choose drugs that do not rely on the kidney for excretion (e.g., atracurium). Nephrotoxic drugs should be avoided. Diuretics should not be given without careful evaluation of the patient's volaemic status and renal hypoperfusion from inadequate intravascular volume should be avoided.

2.1.8 Off-Pump Cardiac Surgery – Anandh Balu

Your first patient on the list today is a 70-year-old diabetic man with triple vessel coronary artery disease who has opted to have off-pump coronary artery bypass graft surgery (OPCABG).

What kinds of cardiac surgery can be performed 'off-pump'?

Only coronary artery bypass graft surgery can be performed off bypass as valvular surgery and aortic surgery require a still heart and cannot be performed with a beating heart.

Which patients stand to benefit from this approach and what are the proposed advantages and disadvantages?

Cardiopulmonary bypass is associated with: systemic inflammatory response, coagulopathy, renal dysfunction and delirium as well as other types of neurological dysfunction. There is therefore a rationale for avoiding cardiopulmonary bypass in patients who have a high perioperative risk of these complications. Avoidance of cross-clamping the aorta and minimising aortic manipulation is theoretically thought to reduce stroke risk and neurocognitive dysfunction. Recovery from off-pump surgery is typically a lot quicker and the length of intensive care and inpatient stay is reduced. Disadvantages of off-pump surgery are that there is potentially worse long-term mortality due to incomplete revascularisation or graft occlusion which may be early or late.

What are the major differences in terms of surgical conduct?

For optimal operating conditions, surgeons require a near bloodless field, a relatively still heart for joining anastomoses and myocardial protection. Surgical approach is typically via median sternotomy but can also be performed minimally invasively via anterior left thoracotomy. The latter approach is only used for single vessel grafts so is rarely used. Cardiac stabilisation is achieved using varying devices but causes vertical displacement of the heart and compression of the ventricular walls. Surgical ischaemic preconditioning with clamps has now been largely replaced with the use of intra-coronary shunts to maintain perfusion during coronary grafting. The operation is technically more challenging and requires an experienced surgeon.

How is the conduct of anaesthesia influenced during this type of surgery? What are the concerns?

There are significant haemodynamic alterations during beating heart surgery. Stabilisation can cause displacement of the ventricles above the atria and elevated intracardiac filling pressures. This can lead to distortion of the atrioventricular annuli which leads to functional mitral and tricuspid regurgitation. Clear communication between anaesthetist and surgeon is necessary to mitigate for these changes. Ideally mean arterial pressure (MAP) should be maintained above 70 mmHg and heart rate should be controlled and arrhythmias avoided with correction of electrolytes as required. Trendelenberg positioning can be utilised to maintain venous return. Magnesium 5 g is often given at induction to achieve myocardial stabilisation and reduce arrhythmogenicity. Agents such as esmolol, verapamil and diltiazem have been used to treat tachycardia but esmolol has the advantage of being ultra-fast acting with quick offset. Pacing wires are often utilised intraoperatively. Unlike during cardiac bypass, temperature control is more difficult to achieve and normothermia should be maintained as closely as possible. Transoesophageal echocardiography is challenging in this setting due to displacement and air around the heart, but has become part of the standard monitoring as it can be used to detect early intraoperative ischaemia and regional wall motion abnormalities. Heparinisation is still required during vascular manipulation; however, lower doses are required compared to full bypass as no extracorporeal circuit is required so there is often less coagulopathy. There may still be a need to urgently convert to cardiopulmonary

bypass in the event of sustained ventricular fibrillation or cardiovascular collapse and the risk of conversion is estimated at between 1–5%. Some centres advocate the use of regional techniques such as parasternal blocks, intrathecal opiates or thoracic epidurals to facilitate enhanced recovery in these patients.

Why aren't all CABG operations performed off-pump if it is less invasive?

There are no absolute contraindications to OPCABG; however, caution is advised in patients with severe left ventricular dysfunction, severe valvular insufficiency or uncontrolled or frequent arrhythmias. While short-term mortality following off-pump surgery is comparable to on-pump CABG operations, the long-term randomised control trial (RCT) data shows there are higher rates of graft occlusion and need for revascularisation in off-pump patients. Secondary outcome measures such as stroke risk, perioperative complications, and renal failure were similar across both groups; however, there does seem to be a clear benefit to the high-risk cardiac patient in terms of early mortality at 30 days but there is no difference at 1 or 5 year follow-up. This has led to uptake of OPCAB being limited to centres where there is high volume of surgical expertise in the technique.

Further Reading

Chassot P, van der Linden P, Zaugg M, Mueller X, Spahn D. Off-pump coronary artery bypass surgery: Physiology and anaesthetic management. *British Journal of Anaesthesia*. 2004;92(3): 400–413.

Fudulu D, Benedetto U, Pecchinenda G, Chivasso P, Bruno V, Rapetto F et al. Current outcomes of off-pump versus

on-pump coronary artery bypass grafting: Evidence from randomized controlled trials. *Journal of Thoracic Disease*. 2016;8 (S10): S758–S771.

Hett D. Anaesthesia for off-pump coronary artery surgery. *Continuing Education in Anaesthesia Critical Care and Pain*. 2006;6 (2): 60–62.

2.1.9 Intra-aortic Balloon Counterpulsation and Other Assist Devices – Philip Harrington

This question could be presented as part of a short case or as a clinical science question on equipment.

You are looking after a patient on the intensive care unit who has returned from the cardiac catheter laboratory after undergoing investigation and treatment. They have an intra-aortic balloon pump (IABP) in situ.

On what principle does the IABP work?

The intra-aortic balloon pump is a circulatory assist device and works on the principle of synchronised counterpulsation. It supports the coronary and systemic circulation by augmenting cardiac output, decreasing myocardial oxygen demand and increasing the myocardial oxygen supply. This occurs via a balloon, positioned in the descending aorta, that is timed to inflate in diastole and deflate in systole. On inflation, blood displaced proximally increases aortic root pressure and coronary blood flow while blood displaced distally may improve blood flow to more systemic vessels. The deflation of the balloon in

systole reduces afterload leading to improved left ventricular performance and decreased oxygen consumption.

The balloon is inflated with helium and its inflation can be timed via the ECG (with inflation in the middle of the T wave and deflation with the peak of the R-wave) or the arterial pressure waveform detected via the pressure transducer in the case of arrhythmia. Inflation can be timed in a 1:1 fashion with each cardiac cycle or with more extended ratios when weaning is being considered.

What are the indications?

Indications can be related to pathology or following procedures. Decreased myocardial performance following cardiogenic shock of various aetiologies, following acute myocardial infarction, refractory left ventricular failure, unstable angina, acute mitral regurgitation and ventricular septal defects, cardiomyopathy and refractory ventricular arrhythmias are all indications. In addition, weaning from cardiopulmonary bypass, following catheterisation and angioplasty and after cardiac surgery are all post-procedural considerations.

What are its contraindications?

Contraindications may be absolute or relative. Absolute contraindications include aortic regurgitation (due to an increase in the degree of regurgitation), aortic dissection, aortic stents and chronic end-stage disease that is not expected to recover. Relative contradictions consist of aortic aneurysms, severe peripheral vascular disease, major reconstructive surgery, uncontrolled sepsis and tachyarrhythmias rendering synchronisation impossible.

Are you aware of any trials supporting IABP use?

The most recent RCT examining this is the IABP-SHOCK II study published in NEJM in 2012. Patients with acute coronary syndromes and associated cardiogenic shock with planned PCI were randomised to IABP or no IABP. Thirty-day mortality was no different between the two arms of the study and no secondary endpoints in terms of physiology scoring, safety profiles or tissue oxygenation showed differences between the groups. With no statistical difference in safety between the groups the trial did, however, suggest that IABP use is a safe intervention in these deteriorating patients.

What complications may arise from the use of IABP?

Complications may be related to the insertion or placement of the device or simply from the device being in situ. Insertion complications include bleeding and vessel injury which may consist of formation of haematoma, pseudoaneurysm, vessel dissection and perforation.

Position-related complications include blood supply to the bowel and kidneys being compromised if the balloon is positioned too distal in the aorta, and left upper limb or cerebral ischaemia if the balloon is positioned too proximal.

In terms of the device being in situ, helium is used to inflate the balloon due to its low density and rapid balloon inflation; this risks embolus if the balloon ruptures. The presence and use of the device also causes mechanical damage to blood components, this can result in haemolysis and thrombocytopaenia.

What other forms of mechanical cardiovascular assist do you know of?

Ventricular assist devices or VADs are devices used to support the left ventricle (LVAD), right ventricle (RVAD) or both (BiVAD) and are inserted surgically. An LVAD usually connects the left atrium to the ascending aorta while an RVAD typically delivers blood from the right atrium to the pulmonary artery. Newer generation devices utilise a centrifugal pump producing continuous rather than pulsatile flow of blood. They aim to reduce the work required of the heart in acute and chronic heart failure as well as in post-procedural states and can be used as a bridge to cardiac recovery, transplantation or as a destination therapy in itself.

Contraindications to their use include, but are not limited to, biventricular failure over the age of 65, severe extra-cardiac organ failure, concurrent severe sepsis, inadequate psychosocial support and concurrent metastatic malignancy. Specific complications of their use include bleeding from insertion or subsequent coagulopathy, cardiac tamponade, cardiovascular instability and infection.

The final form of mechanical cardiovascular support is extracorporeal membrane oxygenation (ECMO) which would be configured in veno-arterial (VA) fashion in order to support the circulation. VA-ECMO allows the support of cardiovascular and respiratory systems as it bypasses both the heart and lungs. Blood is pumped from the venous system (via drainage of the IVC or right atrium) to the arterial system (returning blood to the ascending aorta or femoral artery) and a gas exchanger oxygenates the blood and removes waste CO₂ prior to delivery to the arterial circulation. The result is decreased cardiac work alongside reduced oxygen consumption of the heart as well as delivering well-oxygenated blood to the systemic circulation. VA-ECMO may be used as a bridge to recovery or a bridge to cardiac transplantation.

Further Reading

Harris P, Kuppurao L. Ventricular assist devices. *British Journal of Anaesthesia*. 2012; 12(3): 145–151.

Krishna M, Zacharowski K. Principles of intra-aortic balloon pump counterpulsation.

British Journal of Anaesthesia. 2009; 9(1) : 24–28.

Thiele H, Zeymer U, Neumann FJ et al. Intraaortic balloon Support for myocardial infarction with cardiogenic shock. *New England Journal of Medicine*. 2012; 367:1287–1296.

2.2.1 Daycase Selection – Alison J Brewer and Philip Harrington

As with many topics this question is likely to start with a generic discussion before moving on to specific points about how you would manage a patient presenting for daycase surgery.

What are the advantages of daycase surgery?

There are many advantages of daycase surgery, and this is one of the reasons why the NHS plan has set a target of 75% of all elective surgery being performed on a daycase basis.

The advantages can be classified into patient and staff/NHS factors:

Advantages for patients:

- Most patients prefer daycase rather than overnight stay
- In the elderly population it has been shown to decrease postoperative cognitive dysfunction
- Ideal for children and elderly patients, with minimal time away from a familiar environment so less psychological disturbance
- Reduced hospital acquired infection
- Early mobilisation reducing the DVT risk
- Reduced risk of cancellation due to emergencies on a designated daycase list

Advantages to staff and the NHS:

- Staff are easier to obtain due to social hours
- Increased economic benefits to the trust as there can be an increased efficiency in a well run daycase service
- Reduced waiting lists
- Increases availability of inpatient beds for major cases

Are there any disadvantages that you can think of?

It is important to carefully assess and have robust guidelines for the selection of patients for daycase surgery. There is also inherently a limitation of some anaesthetic techniques, such as epidural or indwelling catheter techniques for analgesia postoperatively. There also has to be facility for admission of a proportion of patients due to intra and postoperative complications.

What surgery can be performed as a daycase, and what are the important considerations?

There has to be consideration as to what surgery is appropriate to be performed as daycase. It is important to make sure that any surgery can be safely done as a daycase without compromising patient care. The complication rate should be low, with minimal blood loss. There must be adequate control of pain, nausea and vomiting and the patients must be able to eat and drink and mobilise within a reasonable time frame after the surgery is complete. The Department of Health has produced guidelines on daycase surgery, with a list of the cases deemed suitable. Overall the principles stated apply to selection of appropriate daycases.

How are you going to select which patients are appropriate to have daycase surgery?

There are social and medical factors that affect whether a patient can have daycase surgery or not.

Social factors include:

- Willingness on the patients' behalf
- Easy access to a phone in their home environment
- Transport back to the hospital readily available
- A responsible adult who can stay with the patient for at least the first 24 hours postoperatively
- Conditions at home should be compatible with postoperative care with adequate toilet and bathroom facilities
- No more than 60 min travelling time back to the hospital should complications occur
- Patients should have a telephone number to contact in case of emergencies
- Patients should be discharged with an advice sheet explaining what to do in case of complications

Medical factors are more difficult to quantify and in many circumstances clinical judgement needs to be employed. In general patients should be:

- Relatively fit (ASA 1 or 2) patients
- Mobile
- Not morbidly obese
- Patients should be selected according to their physiological status and not their chronological age

Some who fall outside guidelines may particularly benefit and should be discussed on a case-by-case basis, for example stable diabetics. Obesity is not an absolute contraindication for daycase surgery, but there are problems with this population.

What are the problems with treating obese patients in a daycase setting?

There needs to be appropriate resources and facilities for the management of obese patients, and specialist equipment and staff may not be available in the daycase setting. There is an increased risk of respiratory compromise in the obese population and late complications may develop more frequently; therefore a longer hospital stay may be

appropriate in some circumstances. Morbidly obese patients may also provide increased technical challenge to surgeons. BMI, though not the ideal tool for assessing these patients, does give pre-assessment nurses a guide for selection. Some daycase units have a BMI cut-off of 35 for all procedures; however, in many units there is no specific limit set and cases can be assessed on an individual basis.

Are there any other factors you would consider in the patient selection process?

Other general factors to consider include:

- The presence of any previous problems with surgery or anaesthetic
- The type of surgery, considering potential for significant bleeding and or prolonged and difficult surgery
- Length of surgery, although techniques such as TIVA and desflurane, that allow rapid emergence, mean that surgery does not have to be time-limited to be performed as daycase
- Requirement for specialist services such as radiological intervention. This may be a logistical reason why daycase surgery may not be appropriate. In these cases surgery should be planned as an inpatient.

What patient-specific factors would prevent you selecting an individual for daycase surgery?

Classify or die – systems approach works best here, CVS, RS, metabolic, endocrine etc.

Cardiovascular history should be explored with particular attention being paid to exercise tolerance and reserve. If there are signs of heart failure, such as ankle oedema and dyspnoea then these patients are not appropriate for daycase surgery. Also uncontrolled angina or myocardial infarction in the last six months should be an exclusion. Valvular heart disease with symptoms should be excluded for further investigation and quantification. Hypertension is controversial and should be controlled preoperatively in a daycase surgery setting for an elective procedure above 180/110 mmHg. A general practitioner should be able to re-refer the patient directly to the clinic for treatment once this is controlled.

Significant respiratory disease should be searched for and optimised prior to day case surgery. A history of corticosteroid use or intensive care admissions recently would make daycase surgery less appropriate.

Diabetics can generally be done if performed first on the list, allowing time for eating and drinking to be established post-procedure. A sliding scale should be able to be administered if necessary in the day surgery unit.

Significant renal, hepatic or severe central nervous system disease should be sought. Epilepsy is not an absolute contraindication, unless there is poor control. Recent TIA's or cerebrovascular events in the past 6 months may exclude daycase surgery. This population group is also more likely to be anticoagulated and every case should be assessed to see if this could be stopped for surgery. If unable to stop, the surgery should be

performed with the patient admitted on an inpatient basis to allow for appropriate bridging with a heparin infusion.

Other drugs that would make day surgery potentially more complicated and inappropriate for daycase include monoamineoxidase inhibitors and corticosteroids. Excessive alcohol or drug abuse may also exclude day surgery but would require discussion on a case-by-case basis.

What are the important principles of anaesthetic management for daycase surgery?

It is important to perform safe and effective anaesthesia, with rapid emergence and few complications. The patients need to recover rapidly, have no cardiovascular or respiratory effects and be awake, orientated and comfortable. They should not have nausea or vomiting and should be able to drink fluids. Antiemetics and avoidance of nitrous oxide in many circumstances is advisable. Adequate analgesia is vital, and opiate sparing techniques are important. Local anaesthetic techniques and NSAIDs should be used if appropriate.

What considerations are there for managing postoperative pain for daycase surgery?

The key issue that often arises with usual analgesic regimes in the postoperative period that is of particular importance in daycase surgery is nausea and vomiting that may occur with opioids such as morphine. This added to inadequate pain control are among the most common reasons for unplanned overnight admission.

It is therefore important to adopt a multimodal analgesic approach targeting different receptors in analgesic pathways and reducing the overall additive side effects. Paracetamol and non-steroidal anti-inflammatory drugs are widely used in daycase surgery in appropriate patient groups and these along with effective surgical use of local anaesthetic can provide good postoperative analgesia.

Opioids are not contraindicated but their use must be closely considered. Side effects such as constipation and urinary retention as well as sedation and respiratory depression may all lead to longer periods of monitoring being required and a delay to prompt discharge. If opioids are required then shorter-acting drugs such as fentanyl may be more appropriate which also has the benefit of less incidence of nausea and vomiting when compared to morphine.

Other possibilities to consider to allow opioid sparing include the use of alpha 2 agonists such as clonidine and dexmedetomidine, gabapentinoids and also intravenous lidocaine.

Would you consider regional techniques to be appropriate?

Don't be put off by being asked a direct question about your practice – if you can consider the various possible issues and come to a conclusion about what you personally do then it shows you have experience in this area. This will likely either help you pass the question or lead to further discussion to display more knowledge.

Effective peripheral nerve blocks can provide excellent analgesia after surgical procedures at the same time as patients remaining ambulatory and functional. The issue with performing these for daycase surgery is either that they do not last long enough with short-acting local anaesthetics or that they wear off after the patient has been discharged leading to uncontrolled pain at home. While not regularly performed it is theoretically possible to perform peripheral nerve block with a catheter left in situ for further local anaesthetic delivery. This would require robust education and follow-up systems being in place for safety and monitoring purposes as well as well-informed and motivated patients with good support at home.

An appropriate approach may be to perform a peripheral nerve block with good patient education around when to start oral analgesia. This will smooth the transition as the nerve block wears off once the patient has returned home.

Neuraxial anaesthesia via spinal injection is also possible within the daycase population. This often avoids the issues of nausea and vomiting and residual sedation and may ease the transition back to eating and drinking and enteral analgesia. Two issues to consider include residual urinary retention and motor blockade that may delay discharge through patients not being ambulatory. These can be overcome with the use of short-acting intrathecal prilocaine that will provide surgical anaesthesia and analgesia while still allowing a timely same-day discharge.

When should patients be discharged and what criteria should they meet?

Prior to discharge the following criteria should be fulfilled:

- Cardiovascularly stable
- No excessive or continuing blood loss
- Oxygen saturation maintained on room air with normal respiratory parameters
- Pain, nausea and vomiting should be controlled
- Any motor block from regional technique should have worn off
- No impairment of bladder function
- Be mobilising independently

As previously mentioned, the discharge environment must also be considered (adequate social care at home, including access to a phone, bathroom and a responsible adult to care for them for 24 hours). Patients must be informed of the actions to take in an emergency or if complications were to develop and be given contact details and information leaflets to this effect.

What are the common reasons for overnight admission following daycase surgery?

There are many reasons why patients get admitted following day surgery. There are surgical reasons, such as bleeding or a more extensive procedure being performed. There are also anaesthetic reasons such as pain, cardiovascular or respiratory instability and nausea and vomiting. If any of the discharge criteria are not achieved, such as urinary retention, then the patient should also be admitted. The commonest reasons for admission are inadequate pain relief and nausea and vomiting.

Further Reading

Aylin P, Williams S, Jarman B, Bottle A.

Trends in day surgery rates. *British Medical Journal*. 2005;331 (7520):803.

Department of Health – Day Surgery

Operational Guide www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/@dh/@en/documents/digitalasset/dh_4060341.pdf.

NHS Evidence Mini topic review: Anaesthesia for day case surgery www.library.nhs.uk/Theatres/ViewResource.aspx?resID=277694.

Thankan L, Faber P. Pain management in day-case surgery. *BJA Education*. 2015; 15 (4) 180–183 available from: www.bjaed.org/action/showPdf?pii=S2058-5349%2817%2930143-9.

2.3.1 Difficult Airway – Kate Leaper and Jonathan J Gatward

An adult patient for a laparoscopic appendicectomy is next on your list. How do you assess their airway?

I would approach their airway assessment systematically, beginning with history, followed by examination, and then review relevant investigations. I use my assessment to identify potential difficulties in managing their airway, such as risk factors for a difficult intubation or mask ventilation, as well as consider other factors that influence my airway strategy such as risk of aspiration or rapid desaturation on induction.

Firstly, I would take a complete medical history, with particular attention to relevant comorbidities such as gastric reflux, obstructive sleep apnoea, rheumatoid arthritis, ankylosing spondylitis, diabetes, or prior radiation to the face or neck. I enquire about any recent respiratory tract infections which can increase the risk of laryngospasm and bronchospasm. I explore any prior anaesthetic issues, which may be apparent to the patient because of a verbal report from previous anaesthetists, an airway letter, medic alert bracelet, a history of prior awake fiberoptic intubation or dental trauma following intubation. Following this I review any available previous anaesthetic charts for documented airway difficulties and previous Cormack and Lehane grades, bearing in mind that these may change over time and so do not guarantee the same view.

I will then examine the patient, looking for factors that can be associated with difficult airway management such as obesity, pregnancy, a small receding jaw, facial hair, or a short, thick neck. This is followed by a more detailed assessment of their airway including dentition, relative tongue size, and specific bedside airway assessments such as mouth opening, modified Mallampati score, thyromental distance, jaw protrusion, and cervical neck mobility.

Finally, I review relevant investigations that are available, such as previous airway imaging like CT scans or nasendoscopy findings.

Can you further describe the specific bedside airway assessments that you use?

Routinely, I assess mouth opening, modified Mallampati, thyromental distance, jaw protrusion and cervical spine mobility.

As the patient opens their mouth during assessment, I use the opportunity to inspect general dentition as well as the inter-incisor gap. A gap of less than 3 cm may make it

difficult to insert a laryngoscope blade, supraglottic airway or oropharyngeal airway. Mouth opening may be limited due to mechanical or functional reasons, which is important to ascertain as the latter may improve with neuromuscular blockade.

The modified Mallampati score involves visually inspecting the pharyngeal structures with the patient sitting up, their mouth fully open, tongue protruded and without phonating. In a class I view, the faucial pillars, soft palate, and uvula are visible. In a class II view, the faucial pillars and soft palate are visible but the uvula is obscured by the tongue. In a class III view, only the soft palate is visible, and in a class IV view, the soft palate is not visible. Classes III and IV are associated with difficult intubation.

Thyromental distance refers to the distance from the thyroid notch to the mental prominence with the head fully extended. Less than 6.5 cm can be associated with difficult intubation, particularly during direct laryngoscopy as it indicates a small mandibular space to displace the tongue into.

Jaw protrusion is a key assessment of temporomandibular joint mobility and can be assessed using Wilson's scoring. In class A, the lower teeth may be placed in front of the upper teeth. In class B, the lower teeth can only be placed in line with the upper teeth. And in class C, which is associated with difficult intubation, the lower teeth cannot be placed in line with the upper teeth.

Cervical spine mobility can be affected by a range of conditions, such as prior surgical fixation, rheumatoid arthritis, and ankylosing spondylitis. Less than 90 degrees of anterior and posterior flexion is associated with difficult intubation.

How accurate are these tests at predicting difficult intubation?

Individually, each of these bedside assessment tools perform poorly so I use multiple tests in combination to better predict difficulty with airway management. For example, the Mallampati score has poor sensitivity, specificity, and positive predictive value. When used in isolation, it only predicts about 50% of difficult intubations via direct laryngoscopy and yields many false positives.

Do you know any scoring systems for predicting a difficult airway?

There are several scoring systems available, for example the Wilson Risk Sum Score. It is based on a combination of five risk factors with a score from 0 to 2 allocated to each. The risk factors are weight, head and neck movement, receding mandible, jaw movement, and buck teeth. A score of 3 or more predicts 75% of difficult intubations but has a false positive rate of 12%.

Your patient for the laparoscopic appendicectomy is a 38-year-old man who has been on intravenous antibiotics for 24 hours, though remains febrile with a heart rate of 105 beats/min and a blood pressure of 115/70 mmHg. He is usually fit and well, though he recently fractured his jaw and had a mandibular repair with wiring. He cannot recall any anaesthetic issues at the time.

On examination, you note that he has limited mouth opening with an inter-incisor gap of 2 cm. How will you manage his airway for this procedure?

It's important to be systematic here – consider patient, surgical, and anaesthetic factors that influence your decision-making in formulating an airway strategy.

This patient has intra-abdominal sepsis and is at increased risk of aspiration and rapid desaturation on induction of anaesthesia. A rapid sequence induction in this patient would be difficult or impossible with his limited mouth opening given it will be hard to insert airway instruments into his mouth. Even inserting a videolaryngoscope will likely prove difficult, and he would be a poor candidate to have multiple attempts at laryngoscopy given his risk profile. This risk profile, together with the fact that he is due to have laparoscopic surgery, with likely head-down positioning, makes tracheal intubation essential, and excludes the intraoperative use of a supraglottic airway device. Given all of this, I would proceed with an awake fiberoptic nasal intubation as it will provide a secure airway with an endotracheal tube in a manner that bypasses the limited mouth opening, preserves spontaneous ventilation and airway reflexes, with the goal of thereby avoiding hypoxaemia and aspiration.

Can you outline your method for awake intubation?

There are a wide range of techniques available, and you may have limited practical experience so choose one and be prepared to justify this method. The common principle behind the variety of techniques is effective application of local anaesthetic to the airway to ensure it is adequately topicalised before instrumentation.

I use the method outlined in the Difficult Airway Society 2020 guidelines for awake tracheal intubation using a fiberoptic technique.

I gain informed consent from the patient, commence monitoring as per AAGBI standards, and ensure I have secure intravenous access with fluid running. As a part of the consent, I describe the technique, including what they will experience, offering reassurance to put them at ease. I administer intramuscular glycopyrrolate 4 mcg/kg as an anti-sialagogue 1 hour pre-procedure, using this route because it may avoid exacerbating his tachycardia. At procedural time-out, I utilise the Difficult Airway Society checklist to brief my team, ensuring we have an experienced anaesthetic assistant and ideally a second anaesthetist, allocate roles, outline the technique for this procedure, and discuss our plan should failure occur. I then check my equipment to ensure all required is available and functioning.

To provide oxygenation, I apply high-flow nasal prongs at 30 l/min and continue its use throughout the procedure, albeit via only one nostril once the other is being instrumented.

I usually find this procedure is well tolerated without sedation in patients who are well informed, understand the rationale for this technique, and are therefore compliant with following instructions. In this case I would be reluctant to offer sedation given the risk of aspiration and rapid desaturation should apnoea occur.

I would sit the patient up at 45 degrees in front of me and ensure I have good view of the patient monitor, infusion pumps and fiberoptic video screen. I gauge which nostril is more likely to be patent by asking the patient to block one at a time and exhale nasally, spraying this nostril with co-phenylcaine for topicalisation and to reduce the risk of epistaxis. Then I ask the patient to poke out their tongue and topicalise their oropharynx with 10% lidocaine spray. I test the adequacy of effective topicalisation using a soft suction catheter before commencing airway instrumentation and re-apply local anaesthetic if required. This may include spraying the selected nostril with 10% lidocaine and reapplying the high-flow nasal prongs to assist aerosolisation.

I advance the pre-loaded fiberoptic scope and endotracheal tube down the selected nostril, ensuring the bevel will be positioned to face posteriorly once vertical. When I have a view of the cords I pause and spray with 1–2 mL of 2% lidocaine via an epidural catheter to topicalise the vocal cords. I carefully advance the endotracheal tube beyond the vocal cords then perform a two-point check to confirm the position of the tracheal tube which involves visualising the carina via the fiberoptic scope and connecting the circuit and confirming the presence of exhaled CO₂ using waveform capnography. Once confirmed, I induce anaesthesia, inflate the tube cuff, and secure the tube.

Can you describe your method of sedation for awake fiberoptic intubation?

I use a low-dose remifentanyl infusion for its sedative and anti-tussive properties. It also has pharmacokinetic advantages in that it is easily titratable with its rapid onset and offset and has organ-independent metabolism. I use the targeted controlled infusion Minto programme to an effect site concentration of 1 to 3 ng/mL, titrated so the patient can respond normally to verbal commands, has a respiratory rate of 8 to 10 breaths/min and there is no significant effect on cardiovascular function. If a second anaesthetist is available, I allocate them the role of titrating the sedation along with monitoring and managing heart rate and blood pressure.

What is the maximum dose of lidocaine that you would use?

The maximum dose recommended by the Difficult Airway Society guidelines is 9 mg/kg of lean body weight. I calculate this and equate it to number of sprays of the lidocaine concentration being used prior to the procedure, keeping in mind that the surgeon may also require local anaesthetic. My practice is to use the minimum required amount of local anaesthetic to achieve adequate topicalisation as some studies have reported toxicity in doses as low as 6 mg/kg.

How many sprays of 10% lidocaine would equate to the maximum dose in this 70 kg patient?

At 70 kg, he can have 630 mg of lidocaine. Ten per cent lidocaine contains 100 mg per mL, so each 0.1 mL spray contains 10 mg lidocaine. So, this patient's maximum recommended dose would be 63 sprays.

Later that evening you are asked to urgently attend the intensive care unit as they are having trouble intubating a patient. When you arrive, the junior airway doctor is performing direct laryngoscopy and is reporting a Cormack and Lehane Grade IV view. The patient monitor displays a heart rate of 135 beats/min, blood pressure of 160/90 mmHg, and oxygen saturations of 93%. How do you approach this situation?

Consider what difficult airway algorithms and cognitive aids you use in your daily practice and show the examiners that you have a systematic approach to a crisis that prioritises

patient safety. Here we use the vortex approach cognitive aid and the Difficult Airway Society algorithm.

I would immediately introduce myself to the team and ask for the difficult airway trolley to be brought to the bedside urgently and ask to take over the airway. I use the principles of the vortex approach in unexpected difficult intubations, which prioritises alveolar oxygenation and focusses on achieving best attempts at the three upper airway techniques of endotracheal intubation, supraglottic airway device insertion and face mask ventilation to achieve this.

Given the attempt at direct laryngoscopy was unsuccessful, I would use face mask ventilation while I ask for the videolaryngoscope to be sourced and set up. I would optimise the patient's head and neck position and then re-attempt endotracheal intubation using the videolaryngoscope with a standard geometry blade. If I failed to gain a view of the glottis, I would move through the optimisation strategies for endotracheal intubation which include manipulations of the head and neck position, removal or adjustment of cricoid pressure, external laryngeal manipulation, adjustments of the laryngoscope position, suction of any secretions or blood obstructing my view, and consideration of an adjunct such as a blind bougie insertion. I would also ensure that the patient has received adequate neuromuscular blockade. If at this stage I was still unable to gain a view, I would attempt laryngoscopy with a hyperangulated videolaryngoscope, using all the same optimisation strategies as with the standard blade.

Unfortunately, you still do not get a view of the glottis at laryngoscopy and are unable to perform endotracheal intubation. What do you do now?

I would call for senior help, declare to the team that I had completed my best effort at endotracheal intubation by laryngoscopy and that our plan now is to concentrate our efforts on the two remaining upper airway techniques, face mask ventilation and supraglottic airway device insertion. I would usually revert to face mask ventilation first, and in this situation, I would use an oropharyngeal airway, a vice-grip and a 2-person technique. I would also consider a nasopharyngeal airway. At this point I would also ask a member of the team to find and open the emergency cricothyroidotomy kit, as I recognise that I am now in the downward spiral through the vortex towards a 'can't intubate, can't oxygenate' (CICO) situation.

I would go through the vortex optimisation strategies for face mask ventilation, which include manipulation of the head, neck and larynx, easing or removing cricoid pressure, suctioning of the airway, and considering differently sized masks and adjuncts. If this was all unsuccessful, I would attempt to insert a second generation supraglottic airway device, then go through all the same optimisation strategies as for face mask ventilation. While I was going through this process, I would ask a member of the team to lay out all the emergency cricothyroidotomy equipment, ready for me to perform scalpel-bougie cricothyroidotomy if needed.

Let us imagine that you achieve end-tidal CO₂ and stable oxygen saturations with your final attempt at supraglottic airway device insertion. How do you proceed?

The presence of end-tidal CO₂ and stable oxygen saturations means that we have achieved some alveolar oxygen delivery and are in the Green Zone of the vortex cognitive

aid. Successful placement of the supraglottic airway device after failed intubation also puts us in the 'Stop and Think' section of the Difficult Airway Society algorithm. During this time, I would focus on re-oxygenating the patient, optimising haemodynamics, mobilising resources such as extra equipment or personnel, and forming an airway strategy. My strategy would be based on the urgency and complexity of the case, the stability of the airway and oxygen saturations, the feasibility of waking the patient up and the skill set and experience of the team. In this case, waking the patient up is not a viable option. My options from here would be to intubate the patient fibre-optically via the supraglottic airway or to proceed to cricothyroidotomy. If at any stage there was a loss of end-tidal CO₂ and falling oxygen saturations, this would signify that we were back in the vortex and I would proceed to cricothyroidotomy, as by this time, I would have completed best efforts at all three upper airway techniques.

After a few minutes of ventilating the patient via the supraglottic airway, you lose the end-tidal CO₂ and the oxygen saturations start to fall. Despite all your manipulations you cannot resolve this. How do you proceed?

I would declare a CICO situation to the team and ask a team member to take over from me at the upper airway, to continue attempts to oxygenate the patient via face mask or supraglottic airway device, while I perform an emergency cricothyroidotomy using the scalpel-bougie technique as recommended by the Difficult Airway Society.

Can you describe how you will perform this?

I need a number 10 blade scalpel, a bougie, and a cuffed size 6.0 mm endotracheal tube. I would identify the anatomical landmarks by performing a laryngeal handshake. Then I would make a transverse stab incision through the cricothyroid membrane, turn the blade 90 degrees so that the sharp edge points towards the feet and carefully slide the curved tip of the bougie along the blade into the trachea. I gently advance the bougie 10 to 15 cm, remove the scalpel and railroad the lubricated tracheal tube over the bougie into the trachea. I would avoid advancing it excessively to avoid endobronchial intubation. Finally, I would remove the bougie, inflate the cuff, connect to the circuit, and confirm ventilation with capnography.

What would you do if you could not feel the cricothyroid membrane?

I would make an 8 to 10 cm longitudinal incision along the midline of the anterior neck, and using my fingers bluntly dissect the soft tissues apart and expose the larynx to identify the cricothyroid membrane by palpation. Once identified, I would use the same technique I described earlier using a scalpel and bougie.

Are there alternative techniques for cricothyroidotomy?

Yes, there is a needle cricothyroidotomy technique.

If you had performed a needle cricothyroidotomy, what oxygen supply would you connect the cannula to?

I would connect the cannula to a high-pressure oxygen source such as the Rapid-O₂ device or Manujet.

Well done, your cricothyroidotomy was successful and you're able to ventilate the patient through the endotracheal tube. Their oxygen saturations have improved, and everyone in the room breathes a collective sigh of relief.

Further Reading

Ahmad I, El-Boghdadly K, Bhagrath R, Hodzovic I, McNarry AF, Mir F, O'Sullivan EP, Patel A, Stacy M, Vaughan D. Difficult Airway Society Guidelines for Awake Tracheal Intubation (ATI) in Adults. *Anaesthesia*. 2020; 75, 509–528.

Difficult Airway Society 2015 guidelines for management of unanticipated difficult

intubation in adults. Frerk C, Mitchell VS, McNarry AF, Mendonca C, Bhagrath R, Patel A., O'Sullivan EP, Woodall NM, Ahmad I, Difficult Airway Society intubation guidelines working group. *British Journal of Anaesthesia*. 2015; 115 (6): 827–848.

The Vortex Approach. <http://vortexapproach.org>.

2.3.2 Performing a Tracheostomy – James W Pearlman and Jonathan J Gatward

You have been requested by the ICU team to assist in the insertion of a percutaneous tracheostomy. On your long walk to the ICU you start thinking about the tracheostomy process. What are the indications for tracheostomy?

Remember, as always, to categorise if possible.

The indications can be subdivided into those for airway obstruction, those for airway protection and conditions that require prolonged access to the lower airway.

Airway obstruction may be caused by tumours, trauma, surgery, burns, anaphylaxis, and infections, such as epiglottitis.

Airway protection might be necessary during head and neck surgery involving the upper airway and larynx, in neurological or muscle diseases such as Guillain-Barré syndrome, bulbar palsies and head injury.

Prolonged access to the lower airway is most often required in patients with respiratory and neurological conditions who require mechanical ventilation and suctioning for secretion removal. Tracheostomies help to avoid upper airway damage due to prolonged translaryngeal intubation, allow for easier suctioning of secretions and may also facilitate weaning from ventilation, as they decrease work of breathing compared to an oral or nasal tracheal tube. Tracheostomies also allow the patient to be awake during a long wean therefore avoiding the complications of long-term sedation. One trial has found that early tracheostomy (TracMan 2013: day 4 vs day 10) may decrease the number of ventilator days, though there was no significant difference in mortality.

What is the anatomy relevant to tracheostomy insertion?

Make sure you know the anatomy. Try to learn a simple line diagram of the larynx, which you can reproduce quickly and reliably (see Figure 2.3.2).

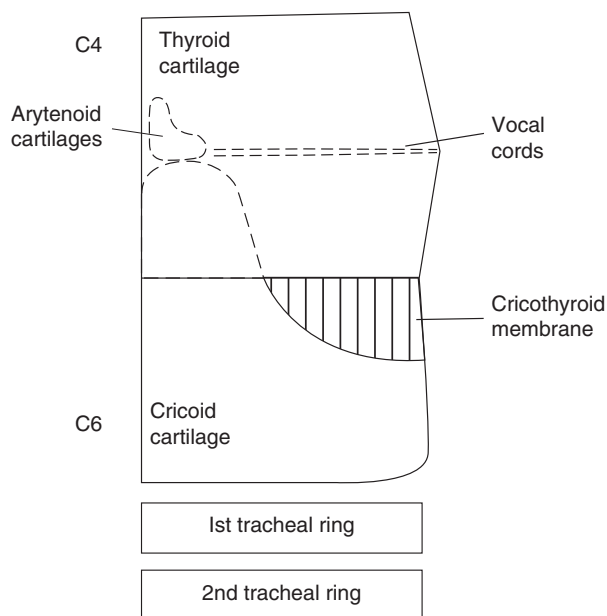


Figure 2.3.2 The larynx.

The larynx lies at the level of the fourth to sixth cervical vertebrae. It is made up of the thyroid, cricoid, arytenoid, cuneiform and corniculate cartilages. The only ones palpable at the front of the neck are the thyroid and cricoid cartilages. The thyroid cartilage is the most prominent laryngeal cartilage, and is usually palpated easily, especially in men. The cricoid lies inferior to this, at the level of C6. It is a signet ring shape, with the narrower band portion lying anteriorly. The cricothyroid membrane is a palpable groove between the lower border of the thyroid cartilage and the upper border of the cricoid cartilage.

Percutaneous tracheostomies are usually inserted between the second and third tracheal rings, as higher approaches may predispose to tracheal stenosis after decannulation. This risk is balanced by the fact that the thyroid isthmus lies between the second and fourth tracheal rings, so there is a risk of damage to this structure causing haemorrhage. Large or aberrant vascular structures can often be identified with ultrasound prior to undertaking the procedure, and the dilation technique will often tamponade any bleeding.

What are some of the techniques for tracheostomy insertion, and can you describe one of them?

There are percutaneous techniques, and a surgical approach. The main percutaneous approaches include the Ciaglia technique, the Griggs technique, and the translaryngeal approach. I will go through the Ciaglia technique.

Given this is an elective procedure, it should be performed in daylight hours at a time when staff are available. An appropriate consent process with the patient or person responsible should be undertaken.

An ideal team would consist of a primary proceduralist, an upper airway operator, a bronchoscopist and at least one other team member to care for the patient and act as a

runner. All members of the team require appropriate PPE including a surgical mask and eye protection.

I would then ensure the patient is fasted, and has vital monitoring on including ECG, oxygen saturations, capnography, and blood pressure. The patient's mouth, pharynx, and trachea should be suctioned. I would then position the patient optimally, with their neck extended with a pillow or a rolled towel beneath the shoulders. I would preoxygenate the patient for 5 minutes prior to performing the procedure. Percutaneous tracheostomy is a sterile procedure, and as such I would surgically scrub, and don a sterile gown and gloves. The tracheostomy equipment and tube need to be checked prior to beginning the procedure. I would then prep the skin with 2% chlorhexidine with alcohol and create a sterile field using drapes. Using the surface anatomy, I would mark the major anatomical landmarks and then use ultrasound to check for any aberrant vascular structures and mark them accordingly.

I would ask the airway operator, using a video laryngoscope, to withdraw the ETT so that the cuff sits just below the vocal cords. The bronchoscopist then introduces the bronchoscope via a perforated angle piece on the ETT so that the trachea and anterior tracheal wall can be visualised. I would then infiltrate 1% lidocaine with adrenaline in the tissue above tracheal rings 2 and 3, and, while aspirating, pierce the trachea in the midline and confirm positioning on bronchoscope. I would then take the airway cannula and a syringe partly filled with sterile 0.9% saline, and slowly introduce the cannula in the midline, aiming posteriorly, perpendicular to the trachea, until air is aspirated, and position confirmed on bronchoscopy. I would advance the cannula under bronchoscope guidance and then remove the needle. I would re-check that air can be aspirated from the cannula, and then insert the guidewire to a depth of 10 cm. I would remove the cannula and confirm the wire placement on bronchoscopy. I would then use a scalpel to create a 1.0–1.5 cm vertical incision with the guidewire in the centre. The short dilator is then inserted until its main body is visible in the trachea. This is followed by the large, graduated dilator, which is inserted by pushing, following its curve, with a constant, controlled force. The dilator should be inserted to the depth marking that corresponds to the outer diameter of the tube to be inserted. The tracheostomy tube is then loaded on an introducer and inserted along the guidewire with constant controlled pressure. The introducer and guidewire are then removed. These steps should be performed under bronchoscopic surveillance, and once the tracheostomy tube is in place, its position should also be confirmed bronchoscopically. The cuff should be inflated, an inner cannula inserted, and the ventilation circuit and capnography attached. Ventilation should be confirmed both clinically and with capnography. The oral ETT should then be removed from the patient. I would examine the patient for bilateral chest expansion, check the capnography trace and the oxygen saturations. A dressing and tracheostomy ties or tapes should then be applied.

How is a percutaneous tracheostomy different to a cricothyroidotomy?

A cricothyroidotomy is an emergency airway rescue technique, whereas percutaneous tracheostomy is usually performed electively on patients in the ICU. The anatomy differs in that cricothyroidotomy is performed via the cricothyroid membrane, while

percutaneous tracheostomies are inserted through the anterior tracheal wall between the second and third tracheal rings.

On arrival to the ICU, they have decided to perform a surgical tracheostomy in the operating theatre. What are the potential pitfalls, and how do you ensure that the airway is maintained throughout the operation?

ICU patients are often sick, physiologically unstable and their management complicated. I may not know the patient, so I would request a full handover and make sure I had read the notes, examined the patient and had seen recent pathology results including a blood gas. I would also like to see a recent chest X-ray and make sure I know the optimal size of the tracheal tube for the patient. I would make sure I know the patient's ventilatory requirements and what infusions they are on. I would ensure that I have full monitoring for transfer and take a skilled assistant with me. I would have a transfer bag with all the relevant drugs and equipment for transfer.

Talk me through the operation and your role in maintaining the airway.

Patient positioning involves extending the neck, at which point the ETT can become dislodged. I would stabilise the tube at this point. Once in position, I would administer 100% oxygen. The surgeons dissect down to the trachea, and then make a vertical incision through one of the second to fourth tracheal rings. They then make a flap or hole in the trachea, at which point the oral tracheal tube can be visualised. There is a risk of damage to the cuff during this process, so I would listen for a cuff leak and watch the oxygen saturations. When the surgeons are ready, they ask for the tracheal tube to be withdrawn. I do this slowly and leave some of it within the larynx in case it needs to be reinserted. The tracheostomy tube is inserted. At this point, there will be limited ventilation due to the presence of the tracheostomy tube within the tracheal lumen. The tracheostomy tube cuff is then inflated and the breathing circuit is then swapped onto the tracheostomy tube. As with the percutaneous tracheostomy, I would examine the patient for bilateral chest expansion; check the capnography trace and the oxygen saturations. The responsibility for holding and securing the tracheostomy tube lies with the surgeons, who may place sutures or secure the tube with ties or tapes. I would only remove the oral tube when I was convinced the tracheostomy was sited correctly. If there was any doubt at all I would insert a bronchoscope to check, looking for positioning with respect to the carina.

Do you know what 'stay sutures' are?

These are long sutures placed into the lateral walls of the tracheal orifice during tracheostomy, the ends of which are taped to the patient's neck either side of the tracheostomy tube. They are used to help reinsertion of the tracheostomy tube should it require changing, fall out or become displaced. Pulling on them brings the tracheal orifice closer to the surface of the skin, so that the orifice can be visualised more easily.

The surgical tracheostomy was undertaken successfully, and you transferred the patient back to the ICU. Later that evening, you are called back urgently. The patient is now awake and breathing spontaneously on pressure support ventilation. The ventilator alarms are sounding, there is an obstructed capnography trace on the monitor, and the oxygen saturations are falling. The ICU registrar and nurse are looking at you for help. What do you do next?

I would assess the patient in a stepwise approach and begin to go through the tracheostomy emergency algorithm. Firstly, I would disconnect the ventilator circuit from the tracheostomy tube and assess for air flow by looking, listening, and feeling at the mouth and the tracheostomy. If I was unsure, I would attach a Mapleson C circuit (flow-inflating bag) and capnography and look for movement of the bag and end-tidal CO₂. If air flow is absent or decreased, I then need to assess the tracheostomy patency. I would not attempt to ventilate through the tracheostomy at this point in case the tracheostomy tube had been displaced into subcutaneous tissue, risking surgical emphysema. I would ask the ICU registrar to commence facemask ventilation, with adjuncts if necessary. I would then remove the inner cannula and attempt to pass a suction catheter. If I am not able to pass a suction catheter beyond the length of the tracheostomy tube, I would deflate the cuff and try again. If I still cannot pass the suction catheter, and the patient is not improving, I need to remove the tracheostomy tube. I would then ask an assistant to cover the tracheostomy stoma with a gloved hand and I would take over facemask ventilation. I would use airway adjuncts as required, and if unsuccessful, step up to supraglottic airway insertion then oral tracheal intubation as required. If all of these were unsuccessful, I would attempt ventilation via the stoma using a paediatric face mask or supraglottic airway pressed over the stoma. As a last resort, if all of these were unsuccessful, I would attempt re-intubation of the stoma using a size 6.0 tracheostomy tube or tracheal tube loaded onto a bronchoscope. If at any stage the patient becomes unresponsive, with absent or abnormal respiration, CPR should be commenced and the resuscitation team called, including the ENT team.

You removed the tracheostomy tube and successfully re-intubated the patient orally. Well done.

Further Reading

National Tracheostomy Safety Project.

Emergency Tracheostomy Algorithms and

management. www.tracheostomy.org.uk/healthcare-staff/emergency-care/emergency-algorithm-tracheostomy.

2.3.3 Bleeding Tonsil and Foreign Body – Caroline SG Janes

Part 1 The bleeding tonsil

You are called in the middle of the night by the ENT ST3 – he would like to take a 6-year-old girl to theatre who is bleeding 8 hours post-tonsillectomy. He would like to proceed immediately and says he is starting to make plans to transfer the patient. He says he will meet you in theatre.

What are the key issues with this case?

This case illustrates a number of anaesthetic challenges; it is a paediatric case involving a difficult airway, which will be shared with the surgeon. The child may be haemodynamically compromised and is at risk of pulmonary aspiration as the stomach will be likely to be full of blood. These issues are further complicated by the likely presence of anxious parents.

The extent of blood loss will be difficult to quantify as the child may have swallowed the blood instead of spitting it out – haemodynamic status should therefore be assessed and resuscitation initiated promptly.

Laryngoscopy and intubation will be challenging as the airway may be compromised by oedema and blood. Children have smaller, more reactive airways, which tolerate manipulation less well. They also have a reduced functional residual capacity and can desaturate rapidly. The likelihood of a stomach full of blood further endangers the airway due to the risk of aspiration.

Other issues specific to paediatric anaesthesia include difficult intravenous access, communication difficulties, anxious parents, temperature regulation and altered drug dosages.

How would you respond to the ENT doctor?

I would inform the ENT doctor that the patient should not leave the ward until I had reviewed her and done a thorough preoperative assessment. In these cases there can be substantial occult blood loss and the child's haemodynamic status needs to be assessed carefully. Adequate fluid resuscitation should be carried out prior to induction as children compensate well despite considerable blood loss. I would also need to discuss this case with the duty consultant prior to proceeding and transferring the child to theatre.

What information would you look for during your preoperative assessment?

As already mentioned, the most important issue to determine is the haemodynamic status which I would assess by thorough history, examination and investigations. I would ask the nurses and parents about excessive swallowing that would suggest occult bleeding or vomiting of blood. I would examine the child to assess capillary refill time, level of consciousness and signs of shock such as pallor and cool peripheries. I would review the ward charts for heart rate, blood pressure, respiratory rate and urine output. Hypotension and decreasing consciousness are late signs of hypovolaemic shock in children and would suggest decompensation. Intravenous access should be present but this should not be assumed and it should be checked for. I would also request an urgent full blood count, clotting screen and cross-match. If point-of-care testing is available this should be used to avoid delays waiting for laboratory results.

I would carry out a routine anaesthetic history and review the initial anaesthetic and drug charts. I would specifically look for the size of endotracheal tube used, recorded laryngeal view, analgesia administered and any difficulties encountered during the initial procedure.

The child's heart rate is 120 beats/min, her respiratory rate is 30 beats/min and her blood pressure is 80/40 mmHg. Capillary refill time is 4 seconds, and her extremities are cool to touch. She is lethargic and complains faintly of feeling sick.

How would you resuscitate this child?

Given these vital signs it is clear that the child has suffered substantial blood loss and requires urgent resuscitation. I would discuss this case with the consultant on-call at the earliest opportunity as this case should be carried out by an experienced anaesthetist. The child should be given high-flow oxygen via a face mask if tolerated and intravenous access should be obtained if this has not been done already. An initial crystalloid bolus of 20 ml/kg should be administered and further fluid resuscitation should be guided by clinical signs. The child should be moved to an area with appropriate monitoring – the most suitable place is often the anaesthetic room.

The bleeding needs to be stopped surgically as soon as possible; however, adequate resuscitation needs to have taken place prior to induction of anaesthesia to avoid cardiovascular collapse. Once the full blood count and clotting screen is known, the use of blood products should be considered. The child should be reassessed after each fluid bolus until she is considered stable enough to proceed with induction.

How would you anaesthetise this child?

I would anaesthetise the child in theatre with the help of an experienced assistant, emergency drugs drawn up and with all airway equipment ready. I would want two suction circuits available in case one became blocked by a blood clot. I would prepare a selection of endotracheal tubes – the same size as used previously and half a size smaller. I would have two of each available in case one became blocked with blood clots. The child should be fully monitored according to the AAGBI minimum standards of monitoring. Video laryngoscopy should be set up ready to use with a size 2 paediatric blade.

The two choices of anaesthetic for a bleeding tonsil are a rapid sequence induction or inhalational induction in the left lateral position and head-down position.

The main advantage of an inhalational induction is that spontaneous ventilation is maintained. The head-down position encourages blood in the airway to drain away from the laryngeal inlet. However, this technique can make the airway more challenging and is seldom used.

I am more familiar with a rapid sequence induction in the supine position using propofol and suxamethonium or rocuronium and it is usually the preferred option. This enables a more rapid procurement of a secure airway with a tracheal tube. Fluids should continue to be titrated to response.

At the end of surgery, I would insert a wide-bore orogastric tube to empty the stomach of blood. I would carefully suction the naso/oropharynx under direct laryngoscopy tilting the head to exclude a 'coroner's clot'. I would extubate the patient in the left lateral, head-down position fully awake. Judicious use of analgesia perioperatively may be topped up in the recovery room as required. The neuromuscular blocking agent should be reversed at the end of the procedure.

What postoperative care should the child receive?

The child should go to recovery for an extended stay with close monitoring. A full blood count and clotting screen should be sent postoperatively and any abnormalities corrected appropriately.

What is the difference between a primary and secondary tonsillar bleed?

The incidence of bleeding following a tonsillectomy is 0.5–5%, and it is usually caused by either venous or capillary ooze. A primary bleed occurs in the immediate postoperative period, usually within 24 hours of surgery, and is most commonly caused by poor haemostasis during surgery. A secondary bleed can occur up to 28 days post-tonsillectomy and is usually secondary to infection or less commonly caused by loosened vessel ties or sloughing off of dead tissue.

What is the blood supply to the tonsillar bed?

The tonsils are supplied by the external carotid artery and its branches. The superior pole is supplied by the tonsillar branches of the ascending pharyngeal artery and lesser palatine artery. The inferior pole is supplied by the ascending palatine artery, dorsal lingual artery and the facial artery branches.

Venous drainage of the tonsils goes to the lingual vein, the pharyngeal and tonsillar capsule plexuses.

What risk factors predispose to post-tonsillectomy bleeding?

The surgical technique is the most important determinant of risk of post-tonsillectomy bleeding. The use of diathermy carries three times the risk of postoperative bleeding compared to a more traditional technique using ties and packs. The latter, however, is accompanied by more intraoperative bleeding.

Other risk factors for post-tonsillectomy bleeding include increasing age, being male and any existing coagulopathies.

Part 2 Swallowed or inhaled foreign body (FB)

A 4-year-old boy presents on the emergency list for rigid bronchoscopy to remove an aspirated safety pin. He has been unwell for a week with a temperature and a cough. Mum reports an episode which occurred 2 weeks ago where he coughed and choked for a few minutes but then he settled down. A Chest X-ray shows the safety pin to be in the right main bronchus.

What are the key considerations in this case?

The most important consideration in any airway is whether established or impending airway compromise is present. This will be more common in an acute presentation and where the FB is lodged in the larynx or trachea. Other anaesthetic challenges include a shared airway and the risk of pulmonary aspiration, particularly if the patient is not starved.

Considerations specific to paediatric anaesthesia include difficult intravenous access, communication difficulties, anxious parents, temperature regulation and altered drug dosages.

How can you differentiate between upper and lower airway obstruction? And why is this important?

It is important to differentiate between upper and lower airway obstruction as they are managed differently.

Foreign bodies in the airway can become impacted in three different places:

- **Supraglottic:** The narrowest point of a paediatric airway is at the level of the cricoid ring. A FB lodged here can cause complete airway obstruction and can be fatal if choking basic life support algorithm is not carried out immediately. If a child does present to hospital with a partial obstruction due to a supraglottic FB this should be treated as an airway emergency as complete obstruction can suddenly follow. The child is likely to present acutely with respiratory distress, stridor and drooling. They will be using accessory muscles of respiration and will have intercostal recession.
- **Trachea:** A FB can also become stuck in the mid-trachea. Symptoms will be similar to those with a supraglottic FB. They will be anxious and are often leaning forward. This should also be treated as an emergency with rapid transfer to theatre to remove the FB.
- **Distal Airways:** Foreign bodies that become lodged more distally, often in the right main bronchus tend to present later unless the event was witnessed. The child may present with a persisting cough, bronchospasm and signs of infection. If they are pyrexial or have raised inflammatory markers they will require a course of antibiotics. The oil from a peanut is especially irritant and can cause mucosal oedema and chemical pneumonitis. These cases, although still in need of urgent FB removal do not need to be treated as emergencies.

A FB can also become stuck in the proximal oesophagus. They may then present with dysphagia and drooling. They will need urgent removal with a rigid oesophagoscopy.

A thorough history and examination can help determine the location of the FB. Chest radiographs will also help, especially if the FB is radiolucent. Areas of hyperinflation from air trapping may be seen distal to the site of the foreign body.

What further information would you like about this case prior to proceeding to theatre?

Firstly, I would like to know whether the child is stable and whether there are signs of respiratory distress. If I suspected airway compromise I would attend to the child immediately and request senior help.

However, if the child is stable, I would take a thorough preoperative history and examine the child. I would review the CXR and discuss the case with ENT surgeons to determine the level at which the foreign body has become lodged. I would ensure the child has been assessed and appropriately treated if there are signs of sepsis. I would arrange to take the child to theatre when they are adequately starved and resuscitated.

The child is stable when calmed by his mother. He has no stridor or drooling. He has a heart rate of 130 beats /min and a respiratory rate of 24 breaths/min with an arterial saturation of 95% on air. His temperature is 37.5 °C.

How would you anaesthetise this child?

In this case there is time to delay surgery until the child is treated for sepsis and is sufficiently fasted. I would contact the consultant-on-call and ask for them to come in for the surgery. I would also request the help of an experienced anaesthetic assistant. Prior to induction of anaesthesia, I would ensure the ENT surgeons were immediately available and ready to proceed with the bronchoscopy, or help establish a surgical airway, if

complete respiratory obstruction should ensue. I would check all my equipment prior to induction and ensure all connections are compatible with the rigid bronchoscope.

With lower airway obstruction, the avoidance of bag-mask ventilation and intermittent positive pressure ventilation is less critical than in upper airway obstruction. I would therefore induce anaesthesia with total intravenous anaesthesia (TIVA) with propofol and 1 mcg/kg fentanyl while aiming to preserve spontaneous ventilation.

Following induction, I would perform laryngoscopy and spray the cords with local anaesthetic (lidocaine 4 mg/kg) to decrease stimulation during instrumentation. I would continue to use Sevoflurane for maintenance of anaesthesia with boluses of opiates (up to 2 mcg/kg of fentanyl or alfentanil 20 mcg/kg).

Retrieval of the foreign body can be attempted when a deep enough plane of anaesthesia is achieved. There are various anaesthetic options during retrieval of the foreign body. A T-piece can be connected to a Storz rigid ventilating bronchoscope, which allows delivery of oxygen and volatile to a spontaneously breathing patient. Intermittent positive pressure ventilation can be also delivered with this system if required.

During the procedure clear and concise communication with the surgeons is required throughout. Hypoxia should be managed promptly by stopping the procedure and returning to bag-mask ventilation. The child's vital signs should be continuously monitored and the chest continually observed. The most common problems encountered will be hypertension, tachycardia or ventricular ectopic beats, hypoxia, hypercarbia and coughing.

Once the FB is removed the larynx, trachea and upper airway should be checked and the chest auscultated to check if the tidal volume has improved and the lower airways have re-expanded.

I would administer dexamethasone to decrease the incidence of airway oedema. If not already administered, I would give intravenous paracetamol and intravenous NSAIDS (unless contraindicated). I would be judicious with opiates, as severe postoperative pain is uncommon.

Postoperatively an LMA or Guedel airway can be inserted for the transfer to recovery.

The child should have a prolonged stay in recovery to observe for reactive oedema and monitor respiratory function. The child will most likely need a course of steroids and antibiotics as the obstruction was prolonged. Chest physiotherapy and humidified oxygen and nebulised adrenaline may also be beneficial. If the child shows signs of sepsis they should be monitored in a high dependency environment.

Would the method of anaesthesia and postoperative care differ if there was a foreign body lodged in the upper airways?

If the FB was lodged in the upper airway I would consider the use of an anticholinergic agent as a premedication. This may be helpful in decreasing secretions and vagal tone thus aiding intubation and avoiding bradycardia during instrumentation of the airway.

These cases are very high risk and should always be done by two experienced anaesthetists as well as a trained assistant. In acute airway obstruction loss of muscle tone can precipitate complete obstruction; neuromuscular blockade should therefore be avoided as should positive pressure ventilation which can push the foreign body down further. This can result in complete occlusion, air trapping or in extreme cases a tension

pneumothorax. Jet ventilation with the Sander Injector should not be used for the same reason. Induction of anaesthesia in these cases would therefore best be done with inhalation of sevoflurane in 100% oxygen with the child sitting on their parent's lap rather than an intravenous induction.

The management during the procedure is otherwise similar to that of a FB in the lower airways.

2.3.4 Obstructive Sleep Apnoea – Jade A Loughran and Sarah F Bell

Sleep apnoea is a topic that comes up frequently. You need to be confident with the definition, clinical presentation and anaesthetic management of the disease.

You are presented with a 56-year-old man, an obese smoker, for an elective large inguinal hernia repair. He also says that he suffers from obstructive sleep apnoea, which was diagnosed 2 years ago. He currently uses a CPAP machine overnight.

How common is sleep apnoea?

About 3% of middle-aged adults have clinically significant sleep apnoea with the male:female ratio of 2:1.

What are the causes of sleep apnoea?

Try and classify your answer based on the question.

Sleep apnoea can be caused by either obstructive or central factors. Obstructive sleep apnoea is characterised by persistent respiratory effort without airflow, while in central apnoea the respiratory effort is absent.

Obstructive sleep apnoea is due to pharyngeal collapse that occurs during sleep. Vibration of the flaccid structures causes snoring which persists until sleep is interrupted and muscle tone restored. Obstructive sleep apnoea is associated with obesity, in which increased adipose tissue contributes to narrowing of the airway, so that increased muscle tone is needed to keep the airway patent. Other causes include anatomical pharyngeal or craniofacial abnormalities, tonsillar hypertrophy and conditions such as hypothyroidism and pregnancy. Sedative drugs and alcohol can precipitate the condition.

Central sleep apnoea is due to reduced or absent respiratory effort due to disorders of ventilatory control or neuromuscular function. Patients have reduced ventilatory capacity that is sufficient when awake, but insufficient when asleep (as the respiratory drive decreases and compensatory mechanisms fail). Causes of central sleep apnoea include neuromuscular diseases such as polio or muscular dystrophy, central nervous system problems such as stroke, surgery and head injury, and excessive respiratory load such as kyphoscoliosis.

What are the symptoms of sleep apnoea?

Try not to focus your answer solely on the respiratory symptoms.

Patients with sleep apnoea will give a history suggestive of the condition, describing snoring, restless sleep and excessive daytime tiredness. Collateral history, often from the patients' partner, is very important in describing the pattern of snoring, apnoea episodes and waking. Poor memory, mood changes and headaches may also be

described. Gastro-oesophageal reflux disease, impotence and nocturnal epilepsy are associated with sleep apnoea. Children with sleep apnoea may have behavioural problems and experience frequent respiratory tract infections.

What are the signs of sleep apnoea?

The clinical signs of sleep apnoea may be divided into airway, respiratory, cardiac and other systems. The airway signs include maxillary hypoplasia, retrognathia, nasal obstruction, increased neck circumference (>44 cm), changes to the soft tissues of the palate and decreased oropharyngeal dimensions. The respiratory signs include cyanosis and hypoxia, and bony or muscular chest wall abnormalities. Cardiac signs include hypertension, arrhythmias and right heart failure (raised JVP, pulsatile liver and peripheral oedema). Other systems that might indicate diseases associated with sleep apnoea are the neurological and endocrine systems, with diseases such as stroke, hypothyroidism or acromegaly possibly being present.

And what are the potential consequences of sleep apnoea?

As usual, try and classify your answer!

The sequelae of sleep apnoea can be divided into the different body systems. Neurological consequences include reduced memory and cognition, headaches, anxiety and depression, and intracranial hypertension. Cardiovascular effects include hypertension, ischaemic heart disease, cerebrovascular disease and right heart failure. Respiratory problems include hypoxaemia, hypercapnia and pulmonary hypertension. Long-term sleep apnoea leads to desensitisation of respiratory centres with increased reliance on the hypoxic drive and eventually type two respiratory failure. The haematological changes include polycythaemia. The endocrine changes include impaired glucose tolerance, dyslipidaemia, reductions in growth hormone and testosterone levels. With regards to the gastrointestinal tract the patient is at risk of developing gastro-oesophageal reflux disease. Finally, sleep apnoea is associated with poor wound healing.

How common is obstructive sleep apnoea?

In the general population the prevalence is 5–10%. It is higher in the adult population undergoing surgery.

How is obstructive sleep apnoea diagnosed?

The diagnosis of obstructive sleep apnoea is made after taking a full history and examining the patient. Screening tools such as the STOP-BANG score are simple and help to identify patients at high risk of obstructive sleep apnoea (For the STOP-BANG score patients score one point each for: loud Snoring, excessive Tiredness, Observed apnoea, high blood Pressure, BMI greater than 35 kg/m^2 , Age over 50 years, Neck circumference greater than 40 cm, and male Gender. A score of 5 or more confers a high probability of moderate to severe sleep apnoea). A sleep study with polysomnography is the gold standard for diagnosis. This involves videoing a patient sleeping and recording measurements such as the EEG (to stage the sleep cycle), 12-lead ECG, pulse oximetry, mouth and nasal airflow, chest and abdominal movement, snoring level and apnoea frequency. Apnoeic episodes are described as 10 seconds or more of total

cessation of airflow despite continuation of respiratory effort against a closed glottis. Partial airway obstruction may cause hypopnoea, with airflow reduced to half for 10 seconds or more. Up to 5 apnoea or hypopnoea episodes per hour are considered normal for an adult. But apnoea-hypopnoea indices (AHI) of more than this represent obstructive sleep apnoea, with 5–15 indicating mild, 15–30 moderate and greater than 30 severe sleep apnoea.

What are the treatment options for obstructive sleep apnoea?

Treatment options are dependent on the severity of the condition and the preference of the patient. Initial measures include weight loss, reduction of alcohol or sedative consumption and cessation of smoking. CPAP is the treatment of choice for patients with moderate to severe sleep apnoea, pressures of 5–20 cmH₂O are used. CPAP acts by splinting open the upper airway; it reduces secondary complications and improves mortality rates. More severe cases may require BIPAP devices.

Some cases of obstructive sleep apnoea may require upper airway imaging to guide whether surgical intervention will be of benefit, although weight loss and CPAP are generally more effective treatments. Correction of nasal obstruction, tonsillectomy and even tracheostomy have all been performed for sleep apnoea patients.

What would be the general anaesthetic considerations for this man?

Sleep apnoea poses a number of challenges to the anaesthetist – firstly the altered respiratory drive, secondly the potential difficult ventilation and intubation and thirdly the multisystem effects of the sleep apnoea.

How would you assess the patient?

I would take a full history from the patient and his partner if possible, paying particular attention to the sleep apnoea and any systemic effects that may have occurred. I would also like to discuss the inguinal hernia and the indications for the operation. My examination would focus on assessing the patient's airway, respiratory and cardiovascular systems. I would want to see a recent full blood count and U&E's to assess for polycythaemia and possible effects of antihypertensive treatment on renal function. I would require a 12-lead ECG, looking for evidence of right heart failure and ischaemic heart disease. An echo and sleep studies might be indicated depending on the severity of the patient's condition.

How would you anaesthetise this man?

Try and picture yourself actually seeing and anaesthetising this patient.

I would avoid sedative premedication but would consider prescribing a preoperative antacid. Where possible I would consider regional anaesthesia. This would depend on the extent of surgery. A general anaesthetic may be needed, and muscle relaxation may be required if the inguinal hernia is large. In this case I would plan to intubate the patient if general anaesthesia were required, although the exact technique would depend on the preoperative history and examination. If the airway appeared difficult I would consent the patient for an awake fiberoptic intubation. Patients with sleep apnoea can become harder to mask ventilate once the muscle relaxant has been administered, and have a

higher incidence of difficult intubation and airway complications such as laryngospasm. I would therefore request senior anaesthetic assistance for this case.

Considerations during maintenance of anaesthesia would include judicious use of opiates and any sedative medication, since this might lead to postoperative respiratory failure. I would use regional or local anaesthetic techniques to provide multimodal analgesia wherever possible and so neuraxial or regional block would be options for this case, depending on the extent of the surgery. This would allow reduced use of opiates. I would aim to maintain normothermia, normocapnia, adequate oxygenation and hydration throughout the procedure. I would extubate him wide awake and would have his CPAP machine available for the immediate postoperative period in case required.

What are your concerns for this patient in the postoperative period?

The patient will require his CPAP in the postoperative period. A high dependency or post-anaesthetic care unit bed should be available. The patient should be nursed sitting (not supine) and care should be taken to monitor his oxygen saturations, respiratory rate and conscious level. Analgesia needs to be carefully considered as opioids will increase the risk of respiratory complications.

Patients with sleep apnoea are at higher risk of developing deep vein thrombosis and so I would consider starting low molecular weight heparin early with TED stockings and pneumatic foot pumps.

Further Reading

Hall A. Sleep physiology and the perioperative care of patients with sleep disorders. *BJA Education*. 2014;15(4):167–172.

Martinez G. Obstructive sleep apnoea. *Continuing Education in Anaesthesia Critical Care and Pain*. 2011; 11(1):5–8.

2.3.5 Upper Airway Infections – Jade A Loughran and Sarah F Bell

This topic helps demonstrate to the examiners that you are safe and have a structured approach to managing a potential airway emergency.

You are called to the resuscitation room to see a 3-year-old boy with stridor. His oxygen saturations are 92% on air, his respiratory rate is 40 breaths/minute and he has audible stridor.

Can you tell me what exactly is stridor?

The examiners might ask an unexpected question to start with. Try to stick to what you know and answer the question!

Stridor is the harsh vibratory sound produced when the airway becomes partially obstructed. It is caused by turbulent flow within the respiratory system. The volume of the stridor does not relate to the degree of airway narrowing.

Can you explain to me why children are more likely to develop stridor?

Paediatric patients have smaller diameter upper and lower airways. A small reduction in the airway causes a marked increase in resistance to flow as described by the Hagen-Poiseuille equation.

How would you assess this child with stridor?

Describe your assessment as though you are performing it in real life. This will reassure the examiner that you have seen or thought carefully about this type of situation before and that you appreciate the important considerations in this case.

I would take a history from the parents, examine the patient and initiate interventions as appropriate. The speed of my actions would depend on the severity of the respiratory compromise of the child. The key during assessment is to cause as little disturbance or distress to the child as possible, since crying or agitation may precipitate complete airway obstruction. I would allow the parents to comfort the child to minimise anxiety and stress.

My history would focus on the presenting complaint. Specifically I would want to know the duration and onset of symptoms, whether any previous treatment had been commenced and whether the child had had any previous episodes. Generally I would enquire about the past medical history, drug history, allergies and anaesthetic history including family history. I would also want to know when the child last ate and drank.

In my examination I would assess the child by observing the airway and respiratory system. I would look to see that the airway was patent and listen to any airway sounds such as stridor, wheeze, grunting or gurgling. I would assess the work of breathing by looking at the respiratory rate, whether there was any head bobbing, nasal flaring, sub or intercostal recession or tracheal tug. I would note the position of the child and whether they preferred a sitting position. I would not attempt to lay the child flat. If tolerated, I would attach a pulse oximeter.

What would be your initial actions?

Again, picture yourself in A&E actually in this situation.

I would attempt to give the child supplementary oxygen via face mask or oxygen tubing held nearby, depending on what was tolerated. I would manage the child in a quiet, calm environment with parents there. I would assess the severity of the respiratory compromise by observing the respiratory rate, oxygen saturations, respiratory effort of the child and conscious level. Importantly I would be looking to see whether these parameters improved with my interventions. I would take a full history from the parents in order to try and formulate a possible diagnosis. I would administer nebulised adrenaline, if tolerated, and call for senior ENT and anaesthetic assistance. I would consider obtaining IV access and a baseline temperature reading, but this would depend on the individual child's temperament and the severity of the illness. It is crucial to avoid further upsetting the patient and worsening the respiratory compromise. I would aim to transfer the child to the theatre suite if no improvements were observed.

What would be in your differential diagnosis for a patient with stridor?

The patient may be suffering from an infection. This may be viral, in the case of croup, or bacterial, in the case of epiglottitis or diphtheria. The patient may have a pharyngeal or peritonsillar abscess. The problem may be due to trauma from burns or prior intubations or there may be a foreign body in the airway. Allergies including anaphylaxis and angioneurotic oedema may also cause stridor as may congenital laryngomalacia.

Can you tell me more about croup?

Croup is also known as laryngotracheobronchitis. It is the cause of 80% of stridor in children and is due to Parainfluenza virus, influenza, RSV or rhinovirus. The condition occurs in children of between 6 months to three years old. Only 1% of cases admitted each year require intubation. The infection causes subglottic and tracheal wall epithelial swelling which can progress to respiratory obstruction. It is typically seen a couple of days after an upper respiratory tract infection. The child may present with a barking cough, low-grade fever and increased respiratory effort. There may also be superimposed bacterial infection.

How would you treat croup?

Try to describe treatment in terms of conservative and then surgical interventions.

The treatment is initially conservative. Depending on the progress of the condition definitive airway management is occasionally required. I would commence therapy with humidified oxygen, nebulised adrenaline (5 ml of 1 in 1000) and consider steroids – this could be nebulised budesonide 2 mg or intravenous dexamethasone 0.15 mg/kg which could be given orally or intravenously. The child might need fluid rehydration, antipyretic medication and antibiotics if superimposed bacterial infection is suspected. Nebulised adrenaline has a transient effect so may need to be repeated.

If you needed to intubate a child with croup how would you go about it?

Again, think of yourself in the situation so that you don't forget anything.

Ideally I would intubate the child in theatre with an experienced anaesthetic team and an ENT surgeon present, since there is always the possibility of a 'can't intubate, can't oxygenate' scenario developing. The parents should ideally be with the child until induction of anaesthesia. I would require a range of airway equipment including different sized endotracheal tubes and bougies in preparation for a difficult intubation. I would have oxygen saturation monitoring and perform a gas induction with sevoflurane and oxygen.

How would you perform your gas induction?

In general I would aim to cause as little extra distress to the child as possible. I would have the child positioned either on a parent's knee or on a trolley and would gradually increase the concentration of sevoflurane delivered via the breathing system. Induction may take longer than expected due to the reduced alveolar ventilation. I would maintain spontaneous ventilation throughout induction but give CPAP to help splint open the airways. Once the patient loses consciousness I would ask the parents to leave and obtain IV access. When the patient reaches a deep plane of anaesthesia (with the pupils small and central) I would wait a further few minutes before attempting to intubate with a smaller than predicted oral tracheal tube.

I would aim to keep the patient spontaneously ventilating with CPAP and sedation until a leak developed around the endotracheal tube. Antibiotic therapy might be required depending on the clinical situation. Sometimes the oral tube is changed for a nasotracheal one if the patient spends a long time on the intensive care unit.

And where would you care for this child?

Ideally the child should be managed on a paediatric intensive care unit. If I were in a district general hospital I would contact the regional centre to arrange a transfer via a retrieval team.

Can you now tell me about epiglottitis?

Yes, epiglottitis is a bacterial infection caused predominantly by *haemophilus influenza* B, but also by *Beta haemolytic streptococcus*, *staphylococcus* and *pneumococci*. The disease has reduced in incidence since the introduction of the Hib vaccine. The bacteria infect the epiglottis, aryepiglottis and arytenoids. Epiglottitis tends to affect children aged 2 to 6 years of age.

How would a child present with epiglottitis?

Try and identify the key features that differentiate this from croup.

The child would have an abrupt onset of high fever and sore throat. They would look extremely unwell. Classically the child would have stridor and sit forward drooling. They might also have dysphagia and loss of a spontaneous cough with muffled speech. The condition might be transiently relieved by nebulised adrenaline.

How would you manage a child with epiglottitis?

Epiglottitis is an infective condition that may lead to rapid loss of the airway and/or septic shock. I would perform my assessment at the same time as starting initial treatment. I would also delegate a member of the team to contact the anaesthetic consultant on call and the ENT surgeon on call to alert them of the patient. My assessment would include a general history and specific to the presenting condition. I would also examine the child looking at the respiratory rate, oxygen saturation, respiratory effort and evidence of dehydration.

Depending on the severity of the child's condition the management can be either conservative or surgical. Initial conservative treatment would include oxygen via face mask and nebulised adrenaline if tolerated. Fluid resuscitation and antibiotics are required but may not be initially administered since complete airway obstruction may occur if the child is stressed. The child should be nursed with parents nearby in an attempt to alleviate anxiety.

If there is no improvement with conservative measures then the patient should be transferred to theatre for intubation.

Now let's say you have transferred the child to theatre uneventfully. You are fully prepared for a gas induction and have a consultant anaesthetist and ENT surgeon with you in theatre. What are the potential problems that might occur and how might these differ from the child with croup?

As with the croup patient, the induction of anaesthesia and intubation may be difficult with the risk of developing into a 'can't intubate can't oxygenate' scenario. In epiglottitis

the deterioration of the child may be more rapid. Furthermore, on laryngoscopy a cherry red epiglottis may be seen and the glottic opening may be extremely difficult to visualise – in this instance asking an assistant to press on the chest and looking for bubbles may guide tracheal tube placement.

The child with epiglottitis may develop severe septic shock with cardiovascular instability requiring aggressive fluid resuscitation and even vasopressor or inotropic support. This is less likely in a child with croup, but coexisting bacterial infection can occur and lead to similar complications.

What would be your further management once the airway was secured?

Blood cultures would need to be sent to confirm the organism and broad-spectrum antibiotic should be commenced as soon as possible. The child would need to be nursed in an intensive care environment until a leak around the tube indicated recovery. This would be expected to occur approximately 48 hours after starting antibiotics. Fluid resuscitation may be required depending on the severity of the sepsis.

Can you tell me anything about bacterial tracheitis?

Bacterial tracheitis is usually caused by staphylococcus, haemophilus, streptococcus or neisseria. A preceding upper respiratory tract infection may occur. The patient would present with a rapid onset of serious illness characterised by fever and respiratory distress. Coughing produces copious tracheal secretions and retrosternal pain. The patient would not usually drool but stridor and a hoarse voice are features. The treatment is similar to epiglottitis but initial intubation may lead to complete obstruction due to pus in the endotracheal tube, forcing the clinician to change the tube. Regular suction will be required and possibly bronchoscopy to remove purulent debris from the trachea. The patient may require intubation for around a week and a prolonged course of antibiotics such as cefuroxime. Tracheal stenosis is a late complication.

Finally, what do you know about retropharyngeal abscesses?

A retropharyngeal abscess is a collection of pus in the space between the posterior pharyngeal wall and prevertebral fascia, caused by lymphatic spread of infection from the sinuses, teeth or middle ear. The infective organism is usually a staphylococcus or streptococcus. Children under 6 years old tend to be affected but it can occur in adults.

What would be the clinical features, investigations and management?

The patient may complain of neck swelling, pain and reduced movement. They may have trismus or drooling and usually have a fever. If performed, a lateral neck X-ray may reveal retropharyngeal thickening. Surgical drainage is usually required but intubation may be extremely difficult. An ENT surgeon should be on standby during induction and I would be prepared for a difficult intubation. A gas induction may be suitable in a child or awake fiberoptic intubation in an adult patient. Care should be taken not to rupture the abscess during intubation since the patient is then at risk of aspirating pus. Antibiotics will be needed along with fluids and antipyretics.

Further Reading

Davies I, Jenkins I. Paediatric airway infections.
BJA Education. 2017;17(10):341–345.

2.3.6 Anaesthesia and Facial Fractures – Gary Thomas

What are the most common causes of maxillofacial injury?

The commonest causes of maxillofacial injury are sporting trauma, road traffic accidents, falls, interpersonal assault and work injuries.

How would you classify facial fractures?

There are several main types of maxillofacial fractures; nasal bones, zygomaticomaxillary, orbital, midface (Le-Fort), mandible and frontal bone.

The fracture of one bone may also be associated with fracture of others and damage to underlying structures.

How might a patient with maxillofacial trauma present?

Much is dependent on the site of the fracture, mechanism of the initial trauma and whether it is a blunt or penetrating injury.

The initial assessment should follow the standard Advanced Trauma Life Support (ATLS) guidelines. Important issues to rule out initially are the potential for airway compromise, bleeding, haemodynamic disturbance, and neurological deficit including injury to the eyes. Disfiguring soft tissue swelling, bruising and haemorrhage can mask damage to underlying structures such as teeth, bones and nerves.

What is the most common facial fracture?

The commonest injury is nasal fracture, accounting for nearly 60% of all facial fractures but most will not require an anaesthetic intervention. Following initial assessment and epistaxis control, the patient may need a CT scan and referral to an ENT surgeon. New fractures can be manipulated in the acute setting but are generally left 5–7 days to allow swelling and oedema to subside before definitive surgical reduction is attempted. Occasionally, nasal fractures are not successfully reduced and become difficult to manipulate without sedation or a general anaesthetic. This might be required if there are functional (nasal obstruction) or cosmetic concerns.

A 24-year-old man is scheduled on an ENT list for a manipulation of nasal fractures on the Daycase Unit. The injury was sustained 10-weeks ago during a game of rugby. He has significant nasal deformity. He has had an appropriate anaesthetic assessment and consented for a general anaesthetic.

How would you anaesthetise this patient?

It is very important to find out how long the procedure is likely to take as this will have a bearing on the anaesthetic management. Therefore, a prior discussion with the ENT surgeon regarding the duration of surgery will be required. It may be a quick 'tweak' of

the nasal bones lasting seconds to a more formal approach lasting up to 30 minutes. Either way, there is potential for post-manipulation nasal bleeding.

If the procedure is to last less than 30 seconds, following the establishment of intravenous access the patient is preoxygenated in the supine position. Then a dose of a short-acting opioid and induction dose of propofol is all that is required. Once asleep, the eyelids should be taped down and while the head is being stabilised, the nose can be manipulated. Once the manipulation is completed the patient can quickly be turned into the left lateral position and recovered in a head-down position.

If there is any element of doubt with regard to length of surgery, then a laryngeal mask or definitive airway (oral tracheal tube) and a throat pack should be inserted.

A 20-year-old man presents on your list for the internal fixation of a right zygoma fracture and orbital repair. He sustained the injury two days ago in a fight. He has no other injuries. He has swelling and bruising of the skin over the fracture site.

The zygoma is a bone that articulates with several bones of the craniofacial skeleton comprising the zygomaticomaxillary complex. It acts as a 'shock absorber', dissipating energy away from the base of skull.

Fracture of the zygomatic arch may be nondisplaced, displaced or comminuted.

It is the second most common fracture, usually caused by high impact trauma.

The fracture may involve just the zygomatic arch (type A1), lateral orbital wall (type A2) or infraorbital rim (type A3). More complicated tetrapod fractures (type B and C) require more complex surgery.

What are the implications for the patient?

Apart from the cosmetic effect of facial bone depression, the zygoma forms part of the lateral and inferior walls of the orbit. Therefore, trauma can also result in cranial nerve damage (zygomaticotemporal nerve, V2) leading to paraesthesia in the area of distribution and possible damage to peripheral branches to the facial nerve. Damage to the wall of orbit can lead to alterations in visual acuity, visual fields and ocular eye movements.

How will you manage the anaesthetic?

Having taken a relevant history and examination and satisfied myself that there were no specific contraindications to a standard anaesthetic plan, I would aim to perform a standard intravenous induction with fentanyl and propofol, insert a laryngeal mask airway (LMA) and allow the patient to breathe sevoflurane spontaneously.

Bearing in mind that the head will be under drapes, I would take special care to protect the eye on the uninjured side with tape and padding.

While the surgical team are reducing the fracture and placing the titanium plates into position when retracting the eye, the oculocardiac reflex might be triggered. This can be managed by asking the surgeon to stop retracting the eye and consider administering an intravenous anticholinergic agent (atropine or glycopyrronium).

During surgery, I would administer intravenous paracetamol, diclofenac, dexamethasone and ondansetron to help prevent postoperative nausea and vomiting and analgesia. Further doses of dexamethasone in the postoperative period help reduce swelling and oedema of the operative site.

Classify Le-Fort fractures?

These are often complex fractures of the midface which involve the separation of part or all of the part of the face from the base of the skull. All Le-Fort fracture types involve the pterygoid processes of the sphenoid bones. As a result, there is disruption of the intrinsic buttress system of the midface. Le-Fort fractures can be classified as types I, II, and III (Figures 2.3.6.1–2.3.6.3) depending on the involvement of the zygomatic, nasal and maxillary bones. They are usually caused by significant blunt trauma (falls or road traffic accidents) and are often associated with other head and neck injuries.

The clinical picture and presentation depend on the type of fracture. Le-Fort I and II fractures are typically caused by a force directed directly at the midface, whereas a Le-Fort III fracture is more likely if the force is directed in a downward direction.

A Le-Fort I fracture results from a transverse fracture across the upper maxilla and pterygoid plates of the sphenoid bone just above the floor of the nose separation of the upper maxilla from the hard palate.

There may be swelling of the upper lip with bruising evident on the buccal surface of the mouth. There may be malocclusion and damage to the upper teeth (missing, chipped or loosened).

A Le-Fort II fracture transects the nasal bones and involves the medial-anterior orbital walls, orbital floor, inferior orbital rims and this transversely fractures the posterior maxilla and pterygoid plates. It is often referred to a 'pyramidal' fracture resulting in a 'floating' maxilla.

This type of fracture results in significant deformity, swelling and bruising of the middle of the face and due to nasal fracture, epistaxis and widening of the intercanthal space. Fracture of the orbit can lead to bilateral periorbital oedema, bruising (raccoon eyes) and sensory impairment infraorbitally extending to the upper lip. A mobile maxilla may limit mouth opening and cause dental malocclusion. Bleeding, bruising and swelling of the maxillary atrium and palate have the potential to cause airway problems. There may also be CSF leak from the nose.

A Le-Fort III fracture is the most severe midface injury. It results in cranio-facial separation as the fracture line passes from the nasofrontal area across the medial, posterior and lateral orbital walls, the zygomatic arch, and through the upper portion of pterygoid plates.

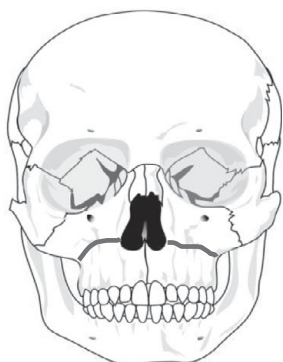


Figure 2.3.6.1 Le-Fort I fracture.

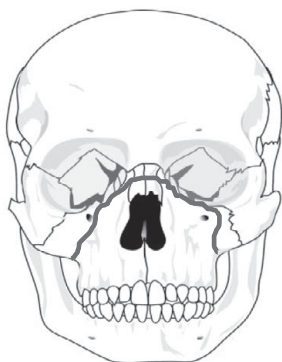


Figure 2.3.6.2 Le-Fort II fracture.

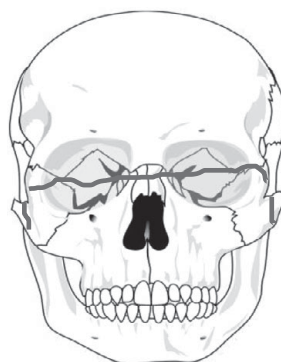


Figure 2.3.6.3 Le-Fort III fracture.

Clinical examination again may reveal bilateral periorbital oedema and bruising (raccoon eyes), ecchymosis of the maxillary buccal atrium and palate, and the face may appear flattened and elongated (dish-face deformity). Examination with an otoscope may reveal bleeding from the middle ear and haemotympanum. There may also be some orbital hooding, enophthalmos and mastoid bruising (Battle's sign). Disruption of the meninges may cause CSF rhinorrhoea and otorrhoea.

A 38-year-old man, victim of a base-ball bat assault, is admitted to the emergency department.

His airway is patent but mouth opening is limited, and there is significant bleeding from his mouth and nose, his oxygen saturation on air is 98%, pulse rate 110 beats/min and blood pressure 165/75 mmHg.

He is conscious and cooperative but appears to have significant midface injuries.

What are the most immediate clinical priorities?

The initial assessment should follow the standard Advanced Trauma Life Support (ATLS) guidelines. The priority is to perform a rapid multidisciplinary clinical assessment and as soon as possible, secure the airway, administer 100% oxygen, and assess the patient's respiratory and cardiovascular status. Large bore intravenous access should also be established.

Prior to definitive clinical and radiological evaluation of the extent of the facial, as well as possible cervical spine and intracranial, injuries the airway should be secured.

What are the options for managing his airway, prior to a CT scan and maxillofacial reconstruction, outlining the advantages and disadvantages of each approach?

1. Rapid sequence induction using fentanyl, propofol and suxamethonium or rocuronium and insertion of a south-facing orotracheal tube or a nasotracheal tube.

Advantages

It is the quickest way of establishing a definitive airway. Propofol reduces the cerebral metabolic rate for oxygen (CMRO₂) and in the event of a failed intubation, rocuronium can be reversed.

Disadvantages

Orotracheal intubation will be potentially difficult because of limited mouth opening, blood and likely swelling in the mouth and disrupted facial anatomy. Even if the airway is patent, face mask ventilation to maintain oxygenation will also be difficult because of distorted, mobile facial anatomy.

In the event of a failed intubation and inability to ventilate and oxygenate the patient, suxamethonium cannot be reversed. Even if this approach was chosen, the front of the

neck would have to be prepared for emergency front of neck access (eFONA), via cricothyroid membrane of percutaneous tracheostomy in advance.

If there were significant traumatic brain injuries, following induction of anaesthesia, propofol and fentanyl have the potential to cause profound hypotension and a fall in cerebral perfusion pressure (CPP). Nasal intubation is contraindicated because of the potential for passing the tube into brain tissue.

2. Awake fibreoptic or videolaryngoscopic oral intubation.

Advantages

Avoids the pitfalls of administering a general anaesthetic to secure the airway.

Disadvantages

There is limited mouth opening, making access difficult. The maxillary fracture could be reduced manually by pulling the maxilla forward and upward resulting in clearing the airway and creating an inter-incisor space, allowing access. The patient might not be cooperative and find it difficult to lie flat. The patient may require sedative medication and there is potential for local anaesthetic toxicity.

3. Elective front of neck access (tracheostomy)

Advantages

Avoids the pitfalls of administering a general anaesthetic to secure the airway. No instrumentation of the mouth and middle of face, having the potential to induce further trauma and bleeding. Can be performed electively in a controlled environment by an experienced ENT surgeon. When the definitive airway has been placed, a general anaesthetic can be administered to allow further assessment and definitive treatment of the facial fractures.

Disadvantages

Patient might not be able to lie flat or be cooperative. May require sedation. Short- and long-term complications of a tracheostomy.

4. Submental intubation

Advantages

A definitive airway can be established without the need for a tracheostomy.

Disadvantages

Difficult to insert without a general anaesthetic.

What are the implications of a frontal bone fracture?

These account for only 5% of facial fractures and usually involve the frontal sinuses.

It is likely that the patient has been injured by a high-energy impact and associated base of skull injuries (cribriform plate plate/CSF leak), intracranial trauma, ophthalmic damage and disrupted sinuses. If the meninges are torn, there is a risk of ascending infection and passing a tracheal or nasogastric tube into the nose risks brain trauma.

Further Reading

Morosan M, Parbhoo A, Curry N. Anaesthesia and common oral and maxillo-facial emergencies. *Continuing Education in Anaesthesia Critical Care and Pain*. 2012; 12(5): 257–262.

Prabhu AJ, Matta BF. Anaesthesia for extra-cranial surgery in patients with traumatic

brain injury. *Continuing Education in Anaesthesia Critical Care and Pain*. 2004; 4(5): 156–159.

Ghabach MB, Abou Rouphael MA, Roumoulian CE, Helou MR. Airway management in a patient with Le-Fort III Fracture. *Saudi Journal of Anaesthesia*. 2014; 128–130.

Enhanced Recovery

2.4.1 Principles of Enhanced Recovery – Ruth Doolin

What is meant by enhanced recovery after surgery?

Enhanced recovery is an evidence-based, standardised, and multidisciplinary perioperative pathway providing guidance for perioperative management.

What are the aims of an enhanced recovery pathway?

The overall aim of enhanced recovery is to help people recover more quickly after having major surgery and to improve the rehabilitation process. By following an enhanced recovery pathway, the patients' pathophysiological and psychological response to surgery can be modified to reduce the stress response during the perioperative period.

Who is involved in the care of patients on an enhanced recovery programme?

The multidisciplinary team involves primarily the patient, supported by GP, surgeons, anaesthetists, nursing staff, physiotherapists, and nutritionists, amongst many others.

What are the benefits of an enhanced recovery programme?

When having major elective surgery, patients partaking in an enhanced recovery programme show a reduction in morbidity and mortality, achieve a quicker return to baseline function and minimise the impact of any complications. Consequently, procedures are more cost-effective due to shorter admissions, fewer readmissions and less demand on critical care. Patient's report increased satisfaction, improved quality of life and improved pain scores, with a resultant reduction in hospital anxiety and depression score indicators.

What specialties do you think particularly benefit from enhanced recovery?

Enhanced recovery was first described in relation to colorectal surgery by Danish surgeon Henrik Kehlet in 1995, but has been expanded to include hepatobiliary, stomach and oesophageal, cardiothoracic, orthopaedic, gynaecological, obstetric, breast and vascular surgeries. More recently the use has also been explored in emergency laparotomy.

Briefly describe the stress response to major surgery.

Injury, or surgery, causes widespread hormonal and metabolic changes, which affect the endocrine, immunological, and haematological systems.

The sympathetic nervous system is activated via the hypothalamus, with a resultant increase in circulating catecholamines. The increased circulating noradrenaline causes tachycardia and hypertension, which are frequently seen intraoperatively. The sympathetic nervous system also affects visceral organ function such as kidney, liver and pancreas through both the catecholamine effect and efferent stimulation.

The hypothalamus also causes an increase in secretion of pituitary hormones. This results in high cortisol and aldosterone levels. The circulating cortisol causes increasing catabolism and mobilisation of energy sources, while the aldosterone promotes salt and water retention. Protein catabolism is enabled primarily by skeletal muscle breakdown, which is then used to generate energy or acute phase proteins. This is often observed by acute muscle wasting and weight loss. Fat metabolism also increases generating fatty acids and glycerol, used as a substrate for gluconeogenesis.

There is also a reduction in insulin and thyroxine levels, and an increase in glucagon and growth hormone secretion. The overall balance tips into insulin resistance. As a result of these changes, the blood glucose concentrations increase, as usual glucose homeostasis is impaired, predisposing to wound infection and impaired healing alongside the usual diabetic complications.

The inflammatory response is mediated by cytokines including interleukins and interferons, produced as an early response to tissue injury. The changes stimulated by cytokines are known as the acute phase response. This is characterised by fevers, granulocytosis, production of acute phase proteins, for example, CRP and fibrinogen, changes in transport proteins, for example, albumin and transferrin, and changes in divalent cations, for example, iron and zinc.

What are the principles of enhanced recovery?

These can be split into preoperative, day of admission, intraoperative and postoperative.

Preoperatively, appropriate patients are ideally identified at the point of referral by GPs and optimised. This includes a particular focus on management of comorbidities, specifically diabetes, hypertension, and anaemia. They should also receive advice and support around diet and exercise, weight optimisation, alcohol and smoking cessation.

On presentation to preoperative clinic, they are risk-stratified to allow informed consent and safe planning of patient care. A significant part of the enhanced recovery programmes is patient education and expectation management, to ensure the patient is motivated and willing to engage with the process. Plans can be made for medicines management including venous thromboembolism (VTE) prophylaxis and anticoagulation, and where appropriate carbohydrate loading with carbohydrate drinks up to 2 hours before, to reduce the insulin resistance associated with the stress response.

Patients should be admitted on the day of surgery. Efforts are made to minimise fasting times, preoperative hypothermia and prolonged immobility while waiting for theatre.

Intraoperatively, the importance of both surgical and anaesthetic techniques should be considered. Surgically, minimally invasive techniques have been shown to reduce complication rate and length of stay. It is recommended to avoid indwelling drains, as

there is limited evidence for benefit, and to remove nasogastric tubes at the end of the procedure where possible as these contribute to atelectasis and postoperative respiratory complications.

Anaesthetically, depth of anaesthesia monitoring should be considered; alternatively, control of MAC is acceptable to avoid overly deep anaesthesia, particularly in elderly patients. In addition, use of neuromuscular blockade monitoring is required to avoid residual paralysis, either by waiting for spontaneous recovery, use of cholinesterase inhibitors or sugammadex to avoid complications, for example, respiratory insufficiency.

Hypothermia should be actively avoided, and glucose should be closely monitored and safely kept close to normal. Normovolaemia should be maintained using goal-directed fluid therapy, aiming to avoid hypoperfusion or tissue oedema. Hypotension is best responded to using vasopressors once normovolaemia is achieved. Management of pain should be opioid sparing and follow a multimodal approach. To avoid post-operative nausea and vomiting prophylactic antiemetic medications are usually given according to individualised risk.

Postoperatively, patients will be cared for in a dedicated specialist recovery area. Any complications (e.g. delirium, postoperative ileus) can be detected early and managed. Early mobilisation is encouraged, with input from physiotherapy and daily targets for ambulation. There is an expectation for early reintroduction of diet and removal of drains, lines, and catheters.

The overall goal is to reduce or minimise the pathophysiological stress caused by the procedure, thereby improving the outcomes.

How would you approach pain management for a patient following an enhanced recovery pathway following surgery?

I would first take a history of their clinical features, impact, and urgency of the procedure, before moving on to their pain history and any preferences they have. Discussion would then focus on exploring and managing their expectations.

I would aim to follow a multimodal approach using the pain ladder as a guide. Options including paracetamol, NSAIDs and opiates are commonly used. Due to the potential adverse side effects of opiates such as nausea and vomiting or paralytic ileus, an opiate sparing approach is preferred.

Regional options should be considered as these can help reduce the need for opiates. For intra-abdominal procedures, commonly used techniques include intrathecal injection or epidural analgesia. Intrathecal injections are often combined with continuous wound infusion of local anaesthetic.

Further adjuncts such as ketamine or gabapentin may also be required for more complex procedures or patients.

How have outcomes from enhanced recovery programmes been studied, and what were the results?

In August 2020 NICE published guideline NG180 titled 'Perioperative care in adults. There is also an evidence review for enhanced recovery programmes.' (National Institute for Health and Care Excellence, 2020) This consisted of a review of 76 randomised controlled trials.

The outcomes found to matter the most included 'health-related quality of life, mortality, patient and family experience, adverse events, complications, and adherence'. Also included were 'length of hospital stay', unplanned intensive care unit admission, length of stay in intensive care unit, hospital readmission and psychological distress and mental well-being'.

The conclusion stated that there was limited evidence for benefit to patients' quality of life and no significant mortality or readmission rate benefit. However, there was a reduction in complication rate, overall pain scores and length of hospital stay.

The review also shows that the enhanced recovery programmes are cost saving, mainly in terms of reduced length of stay and complications

What is required for a successful enhanced recovery programme?

For a successful ERAS programme, a dedicated multidisciplinary team is required, with strong leadership. This leadership could come from the surgical, nursing, or anaesthetic team. Ongoing engagement from all team members, with clear, consistent, and open communication can highlight both success and areas for improvement. Patient engagement through adopting a patient-centred approach, in addition to consistent, understandable information can improve continued compliance. Ongoing audit and quality improvement projects, keeping up to date with emerging evidence and up-to-date research and being receptive to honest feedback keep enhanced recovery programmes running effectively.

Can you think of any potential barriers to a successful enhanced recovery programme?

Barriers could be organisational, for example, a shortage of resources including pre-operative clinic appointments, appropriately trained staff or available beds on the post-operative care unit. In addition, poor compliance to the pathway from either the multidisciplinary team (MDT) or the patient can prevent optimisation of care provided.

Further Reading

- Balfour A, Amery J, Burch, J, Smid – Nanninga H. 'Enhanced recovery after surgery (ERAS®): Barriers and solutions for nurses', *Asia-Pacific Journal of Oncology Nursing*, Elsevier BV. 2022; 9 (7) 100040.
- Desborough JP. 'The stress response to trauma and surgery', *British Journal of Anaesthesia*. 2000; 85 (1) 109–117.
- Feldheiser A, Aziz O, Baldini G, Cox BPBW, Fearon KCH, Feldman LS, Gan, TJ, et al. 'Enhanced recovery after surgery (ERAS) for gastrointestinal surgery, part 2: Consensus statement for anaesthesia practice', *Acta Anaesthesiologica Scandinavica*. 2016; 60 (3), 289–334.
- Ljungqvist O, Hubner M. 'Enhanced recovery after surgery—ERAS—principles, practice and feasibility in the elderly', *Aging Clinical and Experimental Research*. 2018; 30 3, 249–252.
- National Institute for Health and Care Excellence. 2020; *Perioperative Care in Adults [B] Evidence Review for Enhanced Recovery Programmes*.
- NHS.co.uk. 'Enhanced recovery', available at: www.nhs.uk/conditions/enhanced-recovery/ (accessed 28 August 2022).
- NICE. *Perioperative Care in Adults NICE Guideline*. 2020; available at: www.nice.org.uk/guidance/ng180.
- Roulin D, Demartines N. 'Principles of enhanced recovery in gastrointestinal

surgery', *Langenbeck's Archives of Surgery*. 2022.

Sheeran P, Hall GM. 'Cytokines in anaesthesia'. *British Journal of Anaesthesia*. 1997; 78 (2) 201–219.

Tippireddy S, Ghatol D. *Anesthetic Management for Enhanced Recovery after Major Surgery (ERAS)*. 2022.

2.4.2 Minimally Invasive Surgery – Lucinda Williams

What is meant by the term 'minimally invasive surgery'?

Minimally invasive surgery (MIS) is an alternative to open surgery, involving smaller incisions and so less damage to body tissues. MIS includes laparoscopic surgery and robot-assisted surgery. MIS is associated with less pain, greater patient satisfaction, a shorter length of hospital stay and fewer postoperative complications.

What are the disadvantages of MIS?

The main disadvantage of minimally invasive surgery is the loss of haptic, or sensory, feedback for the surgeon. In laparoscopic surgery the haptic feedback is reduced because the instruments have long shafts. In robot-assisted surgery there is an absence of haptic feedback.

Describe the effects of pneumoperitoneum.

During minimally invasive surgery the abdomen is inflated with carbon dioxide in order to create a surgical operating space. CO₂ is the gas of choice because it is inert and does not support combustion if diathermy is used. Due to the fact it is more soluble in blood than nitrogen and oxygen there is less risk of a significant venous embolus should it be inadvertently injected intravascularly.

There are three important principles to consider when managing patients with a pneumoperitoneum:

1. An increase in intra-abdominal pressure
2. Patient positioning: flat, Trendelenburg or reverse Trendelenburg
3. An increase in carbon dioxide absorption.

What effect does an increase in intra-abdominal pressure have?

During laparoscopic surgery the abdominal cavity is inflated with gas. Initially the abdomen and pelvis will expand to accommodate this, but beyond a certain point this compensatory expansion is limited, causing intra-abdominal pressure to increase rapidly.

The effect of increased intra-abdominal pressure is biphasic. Initially, with small increases in pressure (≤ 10 mmHg) there is an increase in venous return and so an increase in cardiac output. However, with increasing pressure in the abdomen (10–20 mmHg) there is compression of the vena cava, which in turn reduces preload to the heart. Compression of the systemic arteries acts to increase systemic vascular resistance. These combine to reduce the cardiac output, which may exacerbate any pre-existing

ventricular failure. To a point the mean arterial pressure is maintained; however, as intra-abdominal pressure increases further (>20 mmHg) the mean arterial pressure starts to drop, as a consequence of the marked reduction in cardiac output.

An increase in intra-abdominal pressure (IAP) causes splinting of the diaphragm. This reduces lung volumes and causes atelectasis, resulting in ventilation-perfusion mismatch. This causes an increase in partial pressure of CO_2 and reduction in the partial pressure of O_2 . These changes correlate with an increased difference in the arterial partial pressure of CO_2 to end-tidal CO_2 ($\text{PaCO}_2 - \text{Pe}'\text{CO}_2$) difference, as well as an increase in the arterial partial pressure of oxygen to fraction of inspired oxygen ($\text{PaO}_2/\text{FiO}_2$) ratio.

Increased IAP is also associated with compromised splanchnic blood flow. Blood flow to the stomach, small and large intestines and liver can be significantly reduced as a consequence of increases in IAP, for example, from 10 to 15 mmHg. This increases the risk of ischaemia-reperfusion injury. There is relative preservation of the retroperitoneal structures; however, the kidneys may be susceptible to reductions in renal blood flow and therefore renal function may be affected. These changes will exacerbate the neuroendocrine effects of surgery, such as activation of the renin-angiotensin-aldosterone-system.

How is physiology influenced by positioning of the patient?

The alterations in physiology seen as a result of pneumoperitoneum are exacerbated by the positioning of the patient during surgery. In the Trendelenburg position lung volumes are reduced, exacerbating atelectasis. There is relative preservation of venous return and cardiac output. In the reverse Trendelenburg position there is greater compromise to cardiovascular function, with a reduction in venous return, cardiac output and very often MAP, with minimal respiratory effects.

Patients undergoing prolonged surgery in the Trendelenburg position, for example, for cystectomy, are at risk of increased intracranial and intraocular pressure, ultimately leading to cerebral oedema. Intravenous fluids should be used judiciously, as they may exacerbate this process.

What effect does CO_2 absorption have?

The peritoneum provides a large surface area for the absorption of carbon dioxide, resulting in an increase in end-tidal CO_2 . This, along with the increase in ventilation-perfusion mismatch caused by pneumoperitoneum, causes a situation where there is an increase in carbon dioxide load combined with reduced carbon dioxide elimination. This may lead to hypercapnia and respiratory acidosis, unless the minute ventilation is increased to compensate for these changes.

What are the principles of preoperative assessment in MIS?

In which pre-existing conditions would MIS be contraindicated?

The suitability of MIS for an individual depends on patient-specific factors and surgery-specific factors. Underpinning the decision is the influence that pneumoperitoneum and positioning will have on the patient's pre-existing medical conditions.

Laparoscopic surgery is largely avoided in patients with valvular heart disease, severe ischaemic heart disease, significant renal dysfunction or advanced respiratory disease.

However, the risk to the individual must be balanced between the likelihood of significant complications vs the shortened recovery time.

There are a number of medical conditions in which MIS is contraindicated.

- Conditions relating to compromised circulation e.g. severe right ventricular failure, biventricular failure or right-to-left cardiac shunt (where the cardiac output (CO) falls as a consequence of the increase in systemic vascular resistance (SVR)) or hypovolaemic shock (where the preload and cardiac output is compromised by the pneumoperitoneum).
- Neurological conditions worsened by an increased IAP e.g. retinal detachment and raised ICP (where the increase in IAP results in an impaired perfusion pressure).

Can you tell me about the anaesthetic management principles of MIS?

The use of pneumoperitoneum necessitates tracheal intubation, with a cuffed oral tracheal tube, in order to protect the patient from aspiration of gastric contents.

Ventilation can be compromised by both Trendelenburg positioning and by pneumoperitoneum, resulting in hypercapnia, respiratory acidosis and increased airway pressures. These changes can be offset to a degree by increasing the minute ventilation; however, a degree of permissive hypercapnia may be necessary to avoid barotrauma. Use of PEEP (positive end-expiratory pressure), pressure-controlled ventilation and low inspiratory pressures can help address ventilation-perfusion mismatch. Nitrous oxide is contraindicated as it causes bowel distension and worsens postoperative nausea and vomiting.

The key principle of fluid management in MIS is maintaining adequate organ perfusion while preventing fluid overload.

Tell me more about the principles of fluid management in MIS.

Enhanced recovery after surgery (ERAS) programmes advocate aiming for a euvolemic state; preoperatively encouraging minimal fasting from clear liquids along with ingestion of carbohydrate-rich drinks. During surgery, fluid management should aim for a neutral-balance, utilising goal-directed therapy where appropriate. There is an emphasis on minimal surgical handling of the bowel which facilitates an early return to usual gut function. Postoperatively, patients are encouraged to eat and drink early, reducing the need for ongoing IV fluids. Cystectomies are notable exceptions, where postoperative ileus is common, and reintroduction of a normal diet may need to be delayed.

What is robot-assisted surgery?

Robot-assisted surgery is a rapidly advancing area of surgery that combines MIS with tele-robotics. Robots do not replace surgeons, rather they are telemanipulators: machines that allow the surgeon to control their surgical instruments from a distance. This allows a greater degree of control and precision as the surgeon is no longer restrained by the anatomy of the human hand, allowing for smaller, more complex movements. Advantages include a shorter hospital stay, reduced postoperative pain scores, faster recovery and return to normal activities.

The most common robot in use in the United Kingdom is the da Vinci system. The surgeon sits away from the patient, at the master console. The console consists of a

stereoscopic eyepiece – which allows the surgeon to view a three-dimensional image of the surgical field – as well as a series of controls and pedals which allow the surgeon to control the robot's arms. The robotic surgical manipulator is made up of three arms which are inserted into the patient via endoscopic ports. The system has the ability to filter and reduce any hand tremor, and actions can be scaled down, allowing the surgeon to perform small, highly complex movements.

What are the disadvantages of robot-assisted surgery?

There are a number of disadvantages of robot-assisted surgery. For the surgeon there is a loss of haptic feedback so they have to rely solely on visual cues to guide their movements. The robot is large and bulky, and once locked into position it can be time-consuming to move, so access to lines and monitoring is difficult for the anaesthetist once the robot is in place. The theatre can feel crowded and cramped, with communication being difficult because the surgeon is sitting away from the patient, in the console. In an emergency or arrest scenario the robot may need to be undocked quickly. To facilitate timely execution of this manoeuvre teams should practise these drills regularly. Robot-assisted surgery can be complex and time-consuming, especially when the team are first familiarising themselves with the process.

Tell me about robot-assisted prostatectomy.

This is the most common procedure that uses robot-assisted techniques. It is associated with a shorter hospital stay, reduced blood loss and lower complication rate than open radical prostatectomy.

What specific considerations are there when anaesthetising patients for this surgery?

The surgery lasts a number of hours, with the patient placed in the lithotomy position with a steep Trendelenburg of 30–45°. Along with this comes the cardiac and respiratory physiological changes already mentioned. Attention must be taken when positioning the patient to ensure excess pressure is not put on vulnerable areas, such as the axillae, elbows, back and shoulders, in order to prevent neuropraxia. Beanbags, straps and gel pads should be used to prevent the patient from slipping. Compartment syndrome (known as 'lithotomy syndrome') may arise as a result of pressure on the calves, thighs or buttocks, and foot pulses should be monitored regularly and compression stockings should be avoided.

Regurgitation of stomach acid during the procedure may result in facial ulceration and conjunctival burns, therefore the eyes should be padded and protected, and the face should be monitored periodically.

The prolonged Trendelenburg results in significant oedema to the head. Postoperatively, patients may be temporarily disoriented and muddled, and they need to be warned of this beforehand. There is a risk of airway oedema and stridor, especially if there has been over judicious fluid administration along with the steep head-down position.

Neurosurgery

2.5.1 Head Injury and Control of ICP – Justin C Mandeville and Menanta van Velze

A 24-year-old motorcyclist is brought into A&E. You attend the trauma call. There is profuse bleeding from the nose and ears and his breathing is noisy. His best response to painful stimulation is to open his eyes, moan, and flex his arms.

What are the important points in the *immediate* management of this patient?

Think of your management of any major trauma patient.

The immediate management of this patient will include a quick, but thorough, primary survey by the trauma team, treating life-threatening conditions as they are found.

Maintenance and protection of the airway with immobilisation of the cervical spine are my first concerns, while excluding catastrophic external haemorrhage. The bleeding nose, noisy breathing and reduced Glasgow Coma Score (GCS) means he will need tracheal intubation to secure his airway. This is complicated, not only by the fact that he is at risk of cervical spine injury, but also cardiovascular instability due to potential concurrent injuries. To maintain his airway, I will start by applying high-flow oxygen, perform a careful jaw thrust and consider using suction, an oropharyngeal airway and assisted ventilation as needed. If not present already, I would ask for an anaesthetic assistant and senior anaesthetic help to be called, while preparing for tracheal intubation by rapid sequence induction (RSI) with manual in-line stabilisation.

In assessing his ventilation, I would count his respiratory rate, measure his oxygen saturations and examine the chest, particularly looking for signs of a haemothorax or pneumothorax, which may need decompressing before positive pressure ventilation was attempted.

I would then assess the circulation by feeling peripheral and central pulses, checking peripheral perfusion and the central capillary refill time in addition to obtaining his heart rate and blood pressure. I would ask for at least two large bore intravenous cannulae to be inserted and bloods to be taken for a blood gas, full blood count, clotting screen, biochemistry, cross-matching of blood and thromboelastography if there is ongoing bleeding or instability. I would simultaneously be considering and assessing for potential sources of haemorrhage.

Next, I would assess neurological function using GCS, which in this case is two for eyes, two for verbal and four for motor – that is eight out of fifteen in total. I would also assess the pupils for size and response.

To complete this systematic ABCDE approach, the patient should be exposed and examined. A trauma CT scan must be performed as soon as possible.

Where able, I would try and obtain information from the ambulance crew, next of kin, GP or hospital notes about allergies, medications, past medical history, the patient's last meal and events leading to the presentation.

How do you categorise the severity of head injury?

You may know more than one way, but this categorisation is widely used.

There are a number of scoring systems, but the most widely used involves the Glasgow Coma Score. The GCS describes levels of motor, verbal and eye responses to defined stimuli and has a minimum score of 3 and maximum score of 15.

A GCS of 13 to 15 on arrival to hospital indicates mild head injury, 9 to 12 indicates moderate and 8 or below would indicate severe injury.

Any patient with a moderate or severe head injury should ideally be managed at a specialist neurosurgical centre.

If time and the examiner would allow, you can go on to explain how GCS is calculated.

What features of a patient with a traumatic brain injury would make you consider intubation of the trachea and ventilation?

There are NICE and AoA Guidelines you may want to refer to here.

Intubation should be considered in any patient with a compromised airway or ventilation. This would include anyone with a GCS of 8 or less, especially if being transferred to a neuroscience unit and particularly prior to the journey if there has been a significant deterioration in conscious level, with an overall fall in GCS by two or more points or a motor score that has fallen by one or more points. Intubation with invasive ventilation is also indicated in any patient with loss of protective airway reflexes, ventilatory insufficiency (as evidenced by hypoxaemia with a PaO_2 of < 13 kPa or hypercarbia with a PaCO_2 of > 6 kPa) or those who are hyperventilating with a PaCO_2 of < 4 kPa.

Other conditions that would indicate intubation are unstable facial fractures, oral or pharyngeal bleeding and seizures.

How would you go about intubating this patient?

The main objectives would be to perform a safe RSI with C-spine protection, while avoiding any hypotension or hypoxia during the process. Having excluded or treated immediate life-threatening injuries, I would use a checklist to ensure all appropriate airway equipment, monitoring and drugs are prepared, that the patient is in a good position, has adequate IV access and is well preoxygenated. I would discuss risks, any airway or other concerns and an intubation plan. My drugs of choice would be fentanyl 3–5 mcg/kg, ketamine 1–2 mg/kg and rocuronium 1.2 mg/kg, with some vasopressors to avoid hypotension. I would use a videolaryngoscope as first line to give me the best chance of successful intubation on the first attempt. After confirmation of endotracheal

tube placement and cardiovascular stability, I would start propofol and alfentanil sedation and apply neuro-protective measures.

You can use any appropriate drug regime, as long as you can justify it.

What is secondary brain injury?

Secondary brain injury is the additional neurological damage that happens after the primary injury or initial insult. It occurs due to localised or generalised physiological upset. Major contributors are hypoxia and hypotension; however, hyper- or hypocapnia, hyper- or hypoglycaemia, pyrexia, seizures, intracranial hypertension and cerebral artery spasm all play a role.

At a cellular level it is thought that the generation of free radicals are probably responsible for cell death. This may be due to what is called 'excitotoxicity', a process in which the excessive release of excitatory neurotransmitters (for example glutamate) cause overstimulation of receptors leading to rising intracellular calcium. The calcium influx activates a number of enzymes, including proteases, phospholipases and endonucleases. These enzymes then go on to damage cell structures such as the cytoskeleton and DNA.

With disruption of the blood-brain barrier and loss of the autoregulation of cerebral blood flow, these problems lead to vasogenic fluid accumulation, a further rise in intracranial pressure (ICP), hypoperfusion, cerebral ischaemia and cerebral hypoxia.

What can be done to prevent secondary brain injury?

In other words, this is asking how you would avoid the causes or contributing factors you just mentioned.

In order to prevent secondary brain injury, I would apply neuroprotective measures in a systematic way to ensure normal physiology as far as possible.

I would ventilate the patient to achieve adequate oxygenation (PaO_2 of more than 13 kPa) and normocapnia (PaCO_2 of 4.5 to 5.0 kPa).

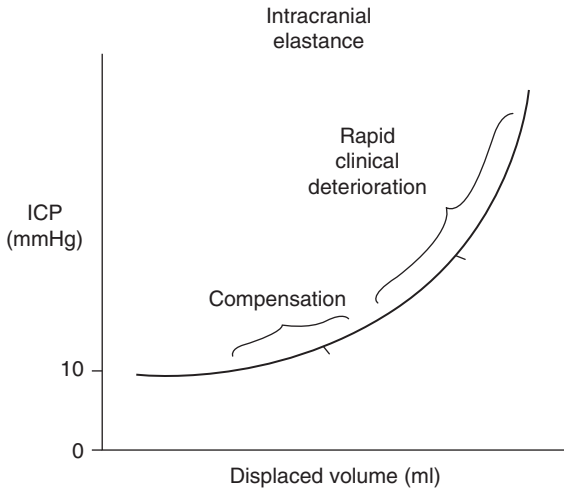
I would avoid hypotension and aim to maintain a cerebral perfusion pressure of 60–70 mmHg. If the ICP is unknown, I would assume that it is equal to 20 mmHg and therefore aim for a mean arterial pressure of >90 mmHg, with a systolic blood pressure of >110 mmHg, but less than 150 mmHg. The transducer should be placed at the level of the tragus.

ICP should be maintained at <20 mmHg. In addition to the already mentioned, I would apply other measures to avoid raised intracranial pressure including to ensure that the patient is positioned head-up at 30 degrees, with the head in a neutral position and no tight ties around the neck. I would ensure adequate sedation and analgesia and consider the need for paralysis. Normoglycaemia and normothermia should be maintained and seizures should be actively treated.

Tell me about the Monro-Kellie Hypothesis.

Draw the elastance curve (Figure 2.5.1).

The Monro-Kellie hypothesis is an explanation of the pressure-volume relationship within the skull. The cranium is basically a rigid box that contains blood, brain tissue and CSF. This means that an increase in the volume of any of these components must be compensated for by the reduction in volume of another or the ICP will rise.

**Figure 2.5.1** Intracranial elastance

Compensation usually occurs due to a reduction in the volume of venous blood, from compression of the veins, and by the movement of CSF out of the cranium into the spinal canal. This means that there's a period of tolerance during which the pressure rises very little in response to changes in volume. When the compensatory mechanisms are overcome the pressure begins to rise sharply; this corresponds to the inflexion point on the elastance curve.

This patient has surgery to relieve an intracranial haemorrhage. Postoperatively, in the intensive care unit, his ICP starts to rise. Briefly tell me of the options available to you for controlling his intracranial hypertension.

Include the options available to you and the surgeons.

I would aim to reduce the ICP to below 20 mmHg, ensuring the ICP transducer is at the level of the tragus and zeroed appropriately. To achieve this, I would categorise my interventions into measures that will decrease brain volume, measures that will decrease cerebral blood volume and measures that will decrease intracranial CSF volume. While optimising these parameters, I would consider the possibility that there may be further intracranial haemorrhage and in discussion with the neurosurgical team, consider CT imaging to check for this.

This question can also easily be answered by classifying your interventions into physical, physiological, pharmacological and surgical options. Some centres classify their management into different stages, for example stage 1, 2 and 3 interventions.

So, how could you decrease blood volume?

Think how you can encourage cerebral venous drainage and reduce cerebral metabolic oxygen requirement and therefore blood flow.

I would optimise venous drainage by sitting the patient up at 30 degrees, ensuring the head is in a neutral position and that there are no ties or collars that could restrict jugular venous flow. I would ensure the patient is ventilated with the minimum PEEP required to maintain good oxygenation and exclude pathologies such as a pneumothorax.

I would ensure there is no hypercapnia and aim for PaCO₂ of 4.5–5 kPa. Ventilating the PaCO₂ down to 4 kPa is not routinely recommended.

Cerebral metabolic oxygen requirement can be reduced with adequate sedation and analgesia, usually in the form of propofol and an opioid, such as alfentanil, but midazolam can be added as a third line drug. With adequate sedation on board, muscle paralysis will avoid coughing and straining. As a last resort, a thiopentone bolus and infusion can be considered with EEG monitoring.

It is also important to avoid pyrexia and treat any seizures. Cooling is not recommended and especially not below 35 degrees Celsius (as per the Eurotherm trial).

Blood in the form of a haematoma may need surgical drainage.

And how can you reduce brain volume?

A decrease in the brain volume is often attained by osmotherapy. Commonly used agents are 0.5 g/kg or 2.5 ml/kg of 20% mannitol or 2–5 ml/kg of 3% NaCl. This can be repeated as required to achieve the desired effect or up to a plasma sodium of 155 mmol/L or a plasma osmolarity of around 310 to 320 mOsm/L.

And to reduce CSF volume?

There is little that can be done to decrease CSF production; however, CSF diversions in the form of an external ventricular drain or lumbar drain may be appropriate to reduce the volume.

Surgical decompression in the form of a decompressive craniectomy should also be considered.

Further Reading

Association of Anaesthetists and the Neuro Anaesthesia and Critical Care Society. Guideline for the safe transfer of the brain-injured patient: Trauma and stroke. 2019.

Head Injury Assessment and Early Management NICE guideline

CG176.2014. Last updated 13 September 2019.

Dinsmore J. Traumatic brain injury: An evidenced-based review of management. *Continuing Education in Anaesthesia Critical Care and Pain*. 2013; 13: 189–195.

2.5.2 Principals of Craniotomy (including Epilepsy Surgery) – Alexandra K Freeman

You have been asked to assess a 55-year-old patient for a craniotomy and debulking of a primary brain tumour.

What are your key considerations in the history when pre-assessing any neurosurgical patient?

A structured approach to the assessment is vital. You must demonstrate this to the examiner.

As with all patients, I would take a thorough medical, surgical, drug and anaesthetic history, considering aspects relevant to any anaesthetic, as well as those specific to the neurosurgical condition. I would establish the presence of any neurological deficit, features of raised intracranial pressure or seizures. Important neurological deficits may

include: motor or sensory disturbance, altered mentation and bulbar dysfunction. I would also assess for any cardiorespiratory or musculoskeletal comorbidities which may influence my anaesthetic technique or affect positioning on the operating table.

Important aspects of the drug history would include the use of steroids (which may require perioperative supplementation), diuretics (which may cause hypovolaemia or electrolyte disturbance), antiepileptics (which may impair liver function or affect the metabolism of other drugs), anticoagulants (which may interfere with surgical haemostasis) and antihypertensives (which may affect haemodynamic stability).

What investigations might you require for this patient?

In addition to any procedure-specific investigations, NICE have published guidelines on 'routine preoperative tests for elective surgery'. It is worth familiarising yourself with these prior to the exam.

The investigations would be guided by my history and examination, as well as any local or national guidelines. They are likely to include a 12-lead ECG, blood tests and brain imaging. The ECG would look for evidence of underlying cardiac abnormalities, arrhythmias or ischaemic changes. A full blood count and clotting studies would look for any coagulation problem which may interfere with surgical haemostasis. Urea and electrolytes may show electrolyte disturbances caused by vomiting, diuretic use or diabetes insipidus. Liver function tests may be deranged from chronic use of anti-epileptic medication. A group and save would be required for all patients; I would request a cross-match if the tumour was highly vascular or in proximity to vascular structures. Imaging studies would be guided by the surgeon and would include CT or MRI scans. If the surgery required use of the sitting position, additional investigations for the presence of a patent foramen ovale (usually a bubble echocardiogram) may be required.

What are your goals for the delivery of anaesthesia for a craniotomy and what anaesthetic technique would you use for this case?

Important goals when providing neuroanaesthesia for craniotomy include:

- Avoiding an increase in intracranial pressure
- Maintaining cerebral perfusion
- Haemodynamic stability
- Allowing early postoperative neurological assessment.

For this case I would use total intravenous anaesthesia with propofol and remifentanyl. Propofol reduces the cerebral metabolic rate and intracranial pressure, while maintaining cerebral perfusion. Remifentanyl is a potent analgesic and is rapidly titratable to periods of noxious stimulation (e.g. intubation and Mayfield pin application), thereby avoiding spikes in intracranial pressure. It also facilitates tube tolerance, avoiding the need for repeated doses of muscle relaxant, which is vital in cases where monitoring of motor evoked potentials are required. Both propofol and remifentanyl have short half-lives resulting in rapid offset, which allows early postoperative neurological assessment. At the time of induction, I would administer a single dose of rocuronium to facilitate intubation, avoiding prolonged hypoventilation, which may cause a deleterious rise in carbon dioxide and intracranial pressure. I would run a metaraminol infusion or have

boluses of vasopressor available to prevent and treat any hypotensive episodes, which may compromise cerebral perfusion.

An alternative technique would be to use a short-acting volatile anaesthetic, such as desflurane or sevoflurane, along with a remifentanyl target controlled infusion. Volatile anaesthetics, however, may increase cerebral blood flow and intracranial pressure. There is also a significant environmental impact from their use. I would avoid the use of nitrous oxide because it increases cerebral metabolic rate and intracranial pressure. It also diffuses into air-filled cavities, which may cause tension pneumocephalus.

You mentioned the use of neuromuscular blocking agents.

Do they affect ICP?

Non-depolarising muscle relaxants do not affect intracranial pressure. The depolarising muscle relaxant, suxamethonium, has been shown to cause a rise in intracranial pressure of 15–20 mmHg. This may, however, be offset by an adequate depth of anaesthesia. Suxamethonium has the advantage of rapid total paralysis, which facilitates prompt airway control and reduces the risk of aspiration. Hypoxia and hypercarbia are more harmful to the brain than the transient elevation in intracranial pressure seen with suxamethonium. An alternative option, if a rapid sequence induction was required, would be to use a rapid-acting dose of rocuronium (0.8–1.2 mg/kg).

What intraoperative monitoring would you require for this patient?

As with any anaesthetic, the patient will require standard AAGBI monitoring, to include: pulse oximetry, capnography, end-tidal volatile anaesthetic concentration (if used), 3-lead ECG, non-invasive blood pressure and a peripheral nerve stimulator. Additional monitoring will include: invasive blood pressure, temperature, urine output, and depth of anaesthesia monitoring (BIS or entropy).

Depending on the location of the tumour, neurophysiological monitoring may be required. This could include electroencephalography, motor and sensory evoked potentials or brainstem auditory evoked potentials. In patients at risk of venous air embolism, central venous pressure monitoring and a precordial Doppler may be used.

What are the commonly used positions for neurosurgery?

Can you tell me the risks and considerations for each?

General considerations relating to all positions include head fixation, difficulty accessing the airway, prolonged immobility, nerve and eye injuries, pressure damage, and risk of dislodging tubes and lines.

The supine position is the most commonly used. A 10-degree head-up tilt may be adopted to improve venous drainage and reduce intracranial pressure. As with all positions, the endotracheal tube should be taped instead of tied to avoid venous congestion. If the supine position is used for surgery to the posterior fossa, the head will be turned to one side risking injury to the brachial plexus; therefore, a sandbag should be placed under the contralateral shoulder.

The prone position is commonly used for surgery to the spinal cord and posterior fossa. During turning of the patient, there is a risk of dislodging tubes and lines. There is

also a risk of awareness from temporary cessation of anaesthetic delivery. Once in the prone position, care must be taken to inspect pressure areas (particularly the breasts, iliac crests, groin and knees). There must be free movement of the abdomen to avoid abdominal compartment syndrome. The neck must be neutral and the eyes must be free from pressure.

The sitting position is used for surgery to the posterior fossa. It provides good surgical access and minimises surgical bleeding but is controversial due to significant risks. These include haemodynamic instability, venous air embolism, tension pneumocephalus, oropharyngeal swelling and quadriplegia. Haemodynamic instability results from venous pooling and should be managed with fluids and vasopressors. Venous air embolism can be catastrophic. Preoperatively, careful patient selection and investigation for the presence of a patent foramen ovale (e.g. with a bubble echocardiogram) can minimise the risk. Intraoperatively, a precordial Doppler and vigilance by the anaesthetist allows early detection. Oropharyngeal swelling and quadriplegia are due to venous congestion from excessive neck flexion, therefore, meticulous attention to neck position is required by both the surgeon and the anaesthetist.

The lateral position is often employed for unilateral craniotomies. The 'park bench' position is a modification of the lateral position, which allows access to midline structures including the spine and posterior fossa, avoiding some of the risks associated with the prone or sitting positions. In both positions, care should be taken to maintain neutrality of the spine and avoid pressure or traction to the arms.

What can you tell me about surgery to the posterior fossa?

The posterior fossa is a rigid compartment with poor compliance. It houses several important structures including the brainstem, cerebellum and cranial nerves. Patients commonly present for surgical management of brain tumours, vascular malformations and cranial nerve pathologies. Posterior fossa surgery is of particular importance to the anaesthetist due to difficulty obtaining surgical access. Positions include prone and sitting, which both carry important risks.

How would you detect and manage a venous air embolism?

Venous air embolism is a medical emergency. Signs may be nonspecific and, therefore, I would maintain a high index of suspicion to any change in physiological parameters. In any patient at risk of venous air embolism, I would consider the use of a precordial Doppler; a change in character and intensity of the sound emitted would indicate intracardiac air, eventually progressing to a mill wheel murmur. Other signs might include a drop in end-tidal CO₂, oxygen desaturation, bronchospasm, hypotension, tachyarrhythmias, ST segment changes, increasing central venous pressure and cardiovascular collapse.

If I suspected the presence of a venous air embolism, I would alert the theatre team and call for help. I would administer 100% oxygen. In order to prevent further entrainment of air, I would tilt the patient head down and request for the surgeons to flood the surgical field with saline. I would then attempt to aspirate air from the right atrium via the central line. Further management is supportive and would include administration of fluids and vasopressors.

What can you tell me about intracerebral aneurysms and how they present?

Intracerebral aneurysms are focal dilatations in the cerebral arteries, which are at risk of spontaneous rupture. They are common and affect around 6% of the population. They usually develop at bifurcations within the Circle of Willis. Risk factors for the development of an aneurysm include hypertension, smoking and genetic predisposition.

They may present with neurological features secondary to their mass effect, subarachnoid haemorrhage following rupture or as an incidental finding. Common features of unruptured aneurysms include headache, visual disturbance, cranial nerve dysfunction, focal neurology or seizures. Features of subarachnoid haemorrhage include a sudden-onset, 'thunderclap' headache, neck stiffness, photophobia, vomiting, decreased level of consciousness, seizures and collapse. Non-neurological features include hypertension, arrhythmias and cardiac dysfunction.

What investigations would be required for a suspected subarachnoid haemorrhage?

The first-line investigation for diagnosis is an urgent non-contrast CT head. If this is negative but there remains a high index of suspicion, a lumbar puncture should be performed to look for xanthochromia.

Following diagnosis of a subarachnoid haemorrhage, a digital subtraction angiogram (DSA) is required to investigate for the presence of an aneurysm and guide surgical management. A CT angiogram (CTA) or magnetic resonance angiogram (MRA) are alternative options; however, they are less sensitive.

What are the definitive treatment options for cerebral aneurysms? What would be your anaesthetic considerations for each?

The definitive treatment options are clipping and coiling. Coiling is the first-line treatment for most aneurysms. It is an endovascular procedure, carried out in the interventional radiology suite. It involves insertion of a catheter, via the femoral artery, into the cerebral circulation, followed by placement of a coil into the aneurysmal sac in order to occlude it. As with any neurosurgical procedure, neuroprotective measures are required. Specific considerations relate to remote site anaesthesia, anticoagulation with IV heparin and the risk of rupture or vascular occlusion. Rupture of the aneurysm can be catastrophic. Management would include reducing blood pressure, reversal of heparin and radiological or surgical control. Conversely, vascular occlusion (due to a thromboembolic event, vasospasm or misplacement of the coil) would require blood pressure augmentation and thrombolysis.

Clipping of an aneurysm requires a craniotomy and is reserved for cases not amenable to coiling, for example those with a wide neck or where coiling has been unsuccessful. The anaesthetic considerations are largely the same as for any craniotomy: avoiding raised ICP, maintaining cerebral perfusion, haemodynamic stability and prompt postoperative neurological assessment. Additionally, increases in transmural pressure must be avoided due to the risk of aneurysm rupture.

When might surgery be implicated in the treatment of epilepsy?

Epilepsy is a common medical condition that is covered elsewhere in this book. It may be useful to review the section on epilepsy (Section 2.5.10) in conjunction with these questions.

Surgery may be considered in patients with epilepsy refractory to medical therapy, with the aim of curing or significantly reducing seizure frequency. Possible surgical procedures include:

- Resection of epileptogenic foci (e.g. anterior temporal lobectomy, hemispherectomy, or resection of brain tumours, vascular malformations or scarring);
- Disconnection surgery (transection of part, or all, of the corpus callosum to prevent propagation of epileptic activity between the hemispheres); and
- Insertion of neuromodulation devices (e.g. vagal nerve stimulators or deep brain stimulators).

What neurological investigations might be carried out prior to epilepsy surgery?

Preoperatively, the patient would undergo investigations to attempt to identify an epileptogenic focus and aid surgical planning. The investigations may include a high-resolution MRI of the brain (to identify an epileptogenic focus) and electroencephalography (for functional localisation of seizures). Functional MRI is also used to localise proximity to eloquent areas and therefore guide surgical planning.

What would be your management if a patient undergoing epilepsy surgery develops a seizure intraoperatively?

Intraoperative seizures are a medical emergency. I would inform the theatre team and call for help. I would administer 100% oxygen and maintain anaesthesia. I would ask the surgeon to irrigate the surgical field with ice-cold saline. If this did not immediately terminate the seizure, I would administer 10–20 mg boluses of propofol. Alternative drug therapy would include benzodiazepines or thiopentone.

Further Reading

Jagannathan S, Krovvidi H. Anaesthetic considerations for posterior fossa surgery. *BJA Education*. 2014; 14(5):202–206.

Larkin CM, O'Brien DF, Maheshwari D. Anaesthesia for epilepsy surgery. *BJA Education* 2019; 19(12):383–389.

National Institute for Health and Care Excellence. Routine preoperative tests for

elective surgery. *NICE*; 2016 [cited 2022 August 18]. (Clinical guideline [NG45]). Available from: www.nice.org.uk/guidance/ng45.

Patel S, Reddy U. Anaesthesia for interventional neuroradiology. *BJA Education*. 2016; 15(5):147–152.

2.5.3 Subarachnoid Haemorrhage – Justin C Mandeville and Philip Harrington

Be aware – some of the nomenclature around subarachnoid haemorrhage has changed in recent years with a good summary provided in the NICE guidance of 2022. While you

would be unlikely to fail by using the term 'vasospasm' (more often being replaced with delayed cerebral ischaemia) the historical use of Triple H therapy has fallen out of favour and should be known about; brief discussions like this with recent evidence make answers very attractive to the examiners!

A 55-year-old woman presents with sudden onset of headache. She is drowsy and confused in the emergency department. CT reveals a subarachnoid haemorrhage (SAH) likely to be from a left anterior cerebral artery aneurysm. You are asked to anaesthetise the patient for angiographic coiling 3 days after this presentation.

What are the important features of the preoperative assessment for this patient?

Essentially normal neuroanaesthesia with potential for catastrophic problems!

This is a procedure generally done in a physiologically stable patient to prevent a more catastrophic second bleed. In contrast to the open approach to an aneurysmal bleed the cranium remains closed so that haemorrhage during the procedure is more dangerous.

At the preoperative visit I would ensure that the patient is not hypovolaemic or overtly hypo- or hypertensive. While specific blood pressure targets have been removed from the NICE guidelines a systolic blood pressure of over 150 mmHg has previously been associated with rebleeding so I would consider managing this if it were the case. I would check for evidence of cardiac ischaemia by requesting an electrocardiogram and I would examine the patient for signs of pulmonary oedema. Nimodipine should have been started as prophylaxis against cerebral vasospasm, and I would check that this had been done. I would perform a neurological assessment to ensure both that the clinical grade of the haemorrhage had not deteriorated and that there were no signs of delayed cerebral ischaemia (DCI). Finally, I would want to identify any concurrent medical problems that may complicate the anaesthetic, particularly pre-existing hypertension, which is common in those with subarachnoid haemorrhage.

In what ways do you think giving this particular anaesthetic differs from most neuroanaesthesia?

Because the cranium is not open, a further bleed may cause a sudden rise in intracranial pressure that the operator cannot deal with directly. Most angiography suites are in a separate part of the hospital from main theatres resulting in remote site anaesthesia, and may produce problems with staffing and equipment. A senior anaesthetist familiar with the surroundings and the procedure should supervise this procedure. In addition, the procedure is not stimulating, which can cause problems with swings in blood pressure, and in particular hypotension.

How would you manage the induction and maintenance of anaesthesia in this case?

Talk about the way you would do it and give reasons for your choices.

I would make sure routine AAGBI monitoring was in place as well as invasive blood pressure monitoring. In addition some units would routinely measure central venous

pressure. A nasopharyngeal or oro-pharyngeal temperature probe should be used, and a urinary catheter should be inserted as the procedure can take several hours. I would make sure that the patient is positioned so that there is no obstruction to flow of jugular venous blood.

My choice would be total intravenous anaesthesia as it is well suited for induction and maintenance of anaesthesia. Hypertension at laryngoscopy is possible and in addition to anaesthetic induction agents I would consider using adjuncts to control this if indicated. I would use rocuronium for muscle relaxation and ensure adequate muscle relaxation throughout the procedure by using a nerve stimulator. I avoid nitrous oxide in these cases for a number of reasons; it may cause cerebral vasodilatation, it may enlarge bubbles that might be inadvertently introduced into the cerebral circulation, and it worsens excitotoxicity. I would utilise ventilator settings that minimise airway pressures and maintain an end-tidal carbon dioxide of 4.0–4.5 kPa.

Vasoactive drugs for control of blood pressure should be readily available. This is important throughout the operation as I may be requested to raise or lower the blood pressure by the operator. Anticoagulation is often used so heparin and protamine should be readily available.

How would you manage this patient postoperatively?

How would you manage them and where?

The most important consideration in the postoperative period is regular neurological assessment looking in particular for the occurrence of delayed cerebral ischaemia. Some centres will monitor all patients on a neurosurgical high dependency unit during the immediate postoperative period; if these are not available then it is also common to manage these patients in the ICU. It is important to manage these patients in conjunction with the neurosurgical team as they may wish to maintain certain blood pressure targets to aid blood flow to potentially ischaemic penumbra. A target of systolic pressure between 140 and 160 mmHg is often used. I would also be vigilant for the complications of subarachnoid haemorrhage.

Which complications would you be mindful of during the procedure?

And how might it affect your anaesthetic?

Intracranial haemorrhage is a possibility during the procedure and may be seen radiologically or may only be noticed when its physiological consequences are seen such as Cushing's sign (hypertension and bradycardia) or cardiac arrhythmias. If it does occur a decision needs to be made as to whether the procedure should be continued, whether the patient should be moved to the neurological intensive care or whether they need an urgent craniotomy. Ischaemia and thrombo-embolic phenomena can occur due to vessel obstruction by the catheter or injectate although there is unlikely to be any obvious sign of this until the patient is woken.

And what complications may occur after the procedure?

Rebleeding after subarachnoid haemorrhage occurs in about 20% of patients within 2 weeks of presentation and 50% within 6 months; if the aneurysm has been secured this is dramatically reduced. The risk of rebleeding is highest within the first 24 hours of onset of symptoms.

Raised intracranial pressure can occur in the absence of a further bleed and may be due to impairment of CSF flow with blood or oedema in the tissue surrounding a haematoma leading to hydrocephalus.

Seizures are also a potential problem both at the time of the bleed and in the ensuing weeks and antiepileptic medication may be needed lifelong.

Groin haematoma is another potential complication and can be worsened by the use of anticoagulants. It may need surgical repair of the vessel defect.

Can you tell me about any scoring systems for subarachnoid haemorrhage?

Scoring systems for SAH may be clinical or radiological.

There are a number of scoring systems, both clinical, including the Hunt and Hess score and the World Federation of Neurosurgeons Score, and radiological, such as the Fisher score.

Pick one of them and outline it for me.

The World Federation of Neurosurgeons Score uses the Glasgow Coma Score and the presence or absence of a focal neurological deficit (Table 2.5.3).

The score relates to both mortality and unfavourable neurological outcome so that grade 1 patients have a mortality of only a few per cent whereas those with grade 5 have significant risk of death or a resulting poor neurological state.

It is important to note that severity grading via SAH scoring systems should not be used *in isolation* to determine requirements for transfer to a neurosurgical centre or how fast this should occur.

What can you tell me about vasospasm?

Vasospasm is an angiographic finding of constricted cerebral arteries. Delayed cerebral ischaemia (DCI) refers to the clinical consequences of neurological deterioration related to ischaemia persisting for over 1 hour and with no other cause. However, the problem is that not all patients with DCI have angiographic vasospasm and vice versa (up to 70% of those with SAH may develop vasospasm but only 30% may have symptoms). Vasospasm is a potential consequence of subarachnoid haemorrhage in which arterial vasoconstriction occurs as a result of irritation by subarachnoid blood. It is generally more likely to happen in those with higher grade haemorrhage and more blood in the subarachnoid space. It usually does not occur until 3 days after the bleed and rarely occurs more than

Table 2.5.3 World Federation of Neurosurgeons classification of subarachnoid haemorrhage

Grade	GCS	Focal neurological deficit
1	15	Absent
2	13–14	Absent
3	13–14	Present
4	7–12	Present or absent
5	<7	Present or absent

2 weeks afterwards. The consequences of vasospasm for the patient can include focal neurological deficit or a more generalised picture with a reduced level of consciousness (DCI) and it is this clinical change that we try to identify and manage.

How do you prevent it?

Any hypovolaemia should be corrected and hydration maintained. Nimodipine 60 mg should be started 4-hourly orally or if this is not possible intravenously. Infusion should start slowly and be titrated to effect and blood pressure. Nimodipine should be continued for 3 weeks. The patient should be regularly checked for focal neurological deficits and if present then urgent imaging should be considered.

How do you treat it?

Treatment should happen in collaboration with the Neurosurgical team.

Firstly it is important to identify DCI and investigate it via a CT scan of the brain to rule out other causes of a change in conscious level in these patients such as rebleed or acute hydrocephalus.

The mainstay of treatment is supportive and used to be via 'triple H therapy' which has now fallen out of favour. It is important to maintain euvolaemia, continue calcium channel blockade as detailed above and discuss with senior neurosurgical decision makers regarding the merits of vasopressors to target a higher blood pressure. It should be noted that improvement in clinical state with vasopressors may be temporary.

Some centres may offer intra-arterial therapies to manage these patients but this would be on a case-by-case basis and currently does not have a robust evidence base.

Other general treatments for critically ill patients with a brain injury should be used including, but not limited to, glucose control, avoidance of fever and sodium homeostasis as well as general critical care measures. The nimodipine should be continued for 1 to 2 weeks once vasospasm has occurred.

Further Reading

Luoma A, Reddy U Acute management of aneurysmal subarachnoid haemorrhage 2013. *Continuing Education in Anaesthesia Critical Care and Pain*. 2013; 13 (2) 52–58.

National Institute of Health and Care Excellence (NICE) *Subarachnoid haemorrhage caused by a ruptured aneurysm: Diagnosis and management* NG228. 2022; available from: www.nice.org.uk/guidance/ng228

2.5.4 Management of Acute Spinal Cord Injury – Carl J Morris and Robert Penders

A 24-year-old man, a motorcyclist, is brought into the emergency department after a high-speed traffic accident, with signs and symptoms compatible with a spinal cord injury around C5.

Describe your initial management of this patient.

Potentially, there's a lot that could be said here – including the entire assessment and management of major trauma. The fact that the examiner specifically mentions spinal cord

injury should alert you to the fact that this is going to be a focus of the structured oral exam. By structuring your answer, and signposting, you avoid wasting time. Your examiner can indicate if they require more details in any area.

I will divide my answer into two parts, firstly looking briefly at the management of serious trauma in general and then addressing the specific management of high spinal cord injury.

The management of any major trauma should follow a structured team-based approach. This would aim to identify and treat life-threatening injuries in a systematic manner. If there is no obvious catastrophic haemorrhage, I would start by assessing the airway (with cervical spine immobilisation to avoid exacerbating cord injury). A decreased level of consciousness, facial or laryngeal injuries might threaten his airway. Initially I would ensure delivery of a high concentration of oxygen via a face mask with a reservoir bag. If there were signs of airway compromise I would proceed to tracheal intubation using a rapid sequence induction with manual in-line stabilisation of the cervical spine.

You might assume these basics are obvious, but if you do not mention them, the examiner may assume you would not do it. The examiner may interrupt during this first section if they are satisfied that you are demonstrating a structured approach to this clinical situation and move on to specific questions about spinal cord injury.

I would then assess and treat injuries threatening ventilation such as an open or tension pneumothorax, massive haemothorax or flail chest. I would move rapidly onto the circulation – securing large bore intravenous access, sending bloods for routine haematology, coagulation, biochemistry and for cross-matching, assessing circulatory adequacy, identifying and where possible attempting to control any major source of haemorrhage, e.g. splinting the pelvis and use of focussed bedside sonography. Haemorrhage control may often require surgical intervention, for example in the case of intra-abdominal, or pelvic bleeding. Resuscitation should ideally be with blood products according to local major haemorrhage protocols. I would also give tranexamic acid in such patients. This would be followed by a neurological assessment, including a Glasgow Coma Score and an attempt to establish the level of any motor or sensory deficit.

With a well-organised team, these activities should happen concurrently, while gathering any relevant history of the event and any medical conditions.

Hopefully, that sort of answer is sufficient to demonstrate that you have experience of major trauma and an overview of the priorities.

To focus in on the spinal cord injury – almost certainly associated with a bony injury to the cervical spine – I will also discuss this in terms of the body systems affected.

With any neck injury there is a risk of haematoma and airway compression. I would have a low threshold for early intubation, with manual immobilisation. With this, I would be mindful of profound bradycardia due to an unopposed vagal response to laryngoscopy. There is also a risk of regurgitation and pulmonary aspiration due to loss of gastro-oesophageal tone.

The degree of acute ventilatory compromise in this patient will be influenced by level of spinal injury. Loss of intercostal muscle function begins at lesions above T8, with complete loss above T1. Patients with higher lesions – especially at and above C3–5 are at particular risk of respiratory compromise due to diaphragmatic dysfunction. It is also possible that the phrenic nerve itself could have been directly injured. This patient is also

at risk of neurogenic pulmonary oedema, due to either the spinal cord injury itself, or an associated head injury. I would monitor respiratory function clinically and with the use of pulse oximetry and arterial blood gases in a critical care environment. Again, I would have a low threshold for intubation and ventilation. I would also bear in mind that with haematoma and cord oedema, the level of the lesion may move cranially in the first 72 hours.

With regards to the cardiovascular system, there is an initial risk of arrhythmia at the time of the cord injury, due to massive sympathetic response and α -adrenergic receptor mediated hypertension. Subsequently, this patient is at risk of neurogenic shock, which would manifest as fluid-resistant hypotension, bradycardia and peripheral vasodilation. I would consider insertion of invasive arterial blood pressure monitoring and early use of anticholinergics and vasopressors in order to maintain cord perfusion pressure and minimise secondary cord injury.

Finally, this patient will require a trauma protocol CT scan. If there were any concerns about potential deterioration in his condition, I would consider elective tracheal intubation and ventilation prior to transfer. This may also be necessary if he were uncooperative due to intoxication, cerebral irritation, or the effects of his other injuries.

How would you intubate this patient if it became necessary?

Here you can demonstrate not just your clinical and technical knowledge, but also awareness of human factors and relevant guidelines associated with difficult airway management.

This is likely to be a difficult intubation due to the combination of cervical immobilisation, associated facial or laryngeal injuries and human and environmental factors.

I would perform a thorough airway assessment and consider performing an awake fibreoptic intubation if there was significant concern about difficult laryngoscopy.

As part of my standard preparations, I would ensure a variety of airway equipment were available. This would include Macintosh and hyperangulated videolaryngoscopes, flexible stylets, gum elastic bougies, a fibreoptic bronchoscope and the means to perform a cricothyroidotomy. I would complete a pre-intubation checklist and follow the Difficult Airway Society guidelines in case of difficulty intubating. I would verbalise my airway management plan, including a plan for failure, with the rest of the team prior to starting.

In this patient, one of the primary goals is to avoid secondary injury to the central nervous system by avoiding hypoxia, hyper- or hypocarbia and maintaining an adequate perfusion pressure to the spinal cord. I would perform a modified rapid sequence induction, with skilled assistants providing two-handed cricoid pressure (to minimise displacement of the cervical spine at C6) and manual in-line immobilisation. I would have vasopressors and atropine immediately available.

There are arguments that spinal injury is a contraindication to cricoid pressure.

Would you have any concerns using suxamethonium?

No, not with a new spinal cord injury. I would be concerned from 48 to 72 hours post-injury though, due to the proliferation of extra-junctional acetylcholine receptors and the associated risk of hyperkalaemia.

What do you mean by neurogenic shock?

Neurogenic shock is a clinical syndrome resulting from the loss of sympathetic output below the spinal cord lesion and subsequent unopposed vagal tone. As I mentioned, this results in hypotension, bradycardia, reduced inotropy and peripheral vasodilation. This is different from 'spinal shock', which refers to a syndrome of flaccid paralysis and loss of motor reflexes initially seen after cord injury and may include neurogenic shock.

How would you manage neurogenic shock?

I would want to avoid over aggressive fluid therapy, which could lead to pulmonary oedema. In this situation I would elect to use vasopressors as required, to maintain sufficient blood pressure for cerebral and renal perfusion. I have mentioned the increased risk of bradycardias or even asystole with manoeuvres that provide vagal stimulation such as intubation or even suction of the tracheal tube. As such I would ensure I have atropine or glycopyrrolate immediately available.

With which level spinal cord injury are these problems associated?

This is seen with lesions at or above T6. More pronounced symptoms are seen where sympathetic cardiac accelerator fibre outputs (between T1 and T5) are disrupted. Neurogenic shock can persist for several weeks post-injury.

What other factors are important in the initial management of this patient?

In the assessment of disability, it is important to remember the high risk of an associated head injury, which in turn would affect airway and ventilatory management. There is likely to be an ileus following the injury and therefore a gastric tube should be inserted early. Temperature regulation will be affected so measures to maintain temperature or to rewarm patients should be instituted. Finally, I would suggest the trauma team liaise with a spinal injuries centre to consider early surgical fixation.

What do you know about the role of corticosteroids?

This is a controversial topic and relies on up-to-date knowledge of recent evidence

Current national guidance from the British Association of Spinal Cord Injury Specialists and NICE state that steroids are not indicated in acute spinal cord injury. This is due to evidence of harm from an increased risk of sepsis, GI bleeding and ARDS.

Are there any other specific problems you anticipate with this patient in the coming months?

As well as the issues already discussed, he will be at risk of gastrointestinal stress ulceration, pressure sores, and in the first few months in particular, at risk of deep vein thrombosis and pulmonary embolism.

Your patient turns out to have an isolated unstable cervical spine injury with a complete transection of the cord. His spinal injury is stabilised in the following few days and he proceeds to rehabilitation.

Can you describe the clinical effects of incomplete transection of the spinal cord, for example anterior spinal cord damage?

This relies on understanding of the anatomy of the spinal cord and relevant motor and sensory tracts and relating it to clinical presentation. Some colourful diagrams should help you remember these different patterns!

The neurological sequelae will reflect the anatomy of the spinal cord and its neural tracts.

Damage to the anterior cord will result in paralysis, due to loss of motor pathways, but proprioception, touch and vibration sense, which are carried in the dorsal columns, will be preserved. This pattern may also be seen with ischaemic damage resulting from the loss of the anterior spinal artery supply to the anterior two thirds of the spinal cord.

In a posterior cord injury, the opposite is seen, with preserved motor power, but loss of touch, vibration and proprioception.

A central cord lesion, causes greater motor loss of the upper limbs than lower, with variable sensory loss and sometimes preservation of bladder and bowel control. This pattern may result from a hyperextension injury.

In a Brown-Sequard lesion – with a hemi-section of the cord – such as may result from a penetrating injury, there is ipsilateral paralysis with loss of proprioception touch and vibration sense and contralateral loss of pain and temperature sensation.

Further Reading

Bonner S, Smith C. Initial management of acute spinal cord injury. *Continuing Education in Anaesthesia Critical Care and Pain*. 2013;13(6): 224–231.

National Institute for Health and Care Excellence (NICE). (2016). *Spinal injury*:

assessment and initial management [NG41]. www.nice.org.uk/guidance/ng4.

Patek M, Stewart M. Spinal cord injury. *Anaesthesia and Intensive Care Medicine*. 2020;21(8): 411–416.

2.5.5 Late Management of Spinal Cord Injury – Carl J Morris and Robert Penders

You are asked to anaesthetise a 58-year-old man for an elective cystoscopy and insertion of a suprapubic catheter, 7 months after complete transection of the cord at C5 level. He gives a history of significant reflux and a history of hypercholesterolaemia.

Please describe your anaesthetic management of the case.

Most anaesthetists will not have direct experience of managing these patients. Therefore an awareness of the key issues should be sufficient for your answer. Questions asking for your ‘anaesthetic management in . . .’ are fairly common in the SOE and you should rehearse answering these using a common structure. By doing this, and by flagging up the key points at the beginning, your examiner is able to relax, already confident in your approach. Also, if time is short you will have already gained crucial marks.

I will divide this into the pre-, peri- and postoperative phases.

The main anaesthetic issues specific to this patient are the risk of autonomic dysreflexia, the potential for a difficult airway, the potential for covert cardiovascular disease

and the risk of suxamethonium-induced hyperkalaemia (if used in a rapid sequence induction).

What do you mean by 'autonomic dysreflexia' (also referred to as 'hyper reflexia')?

This refers to a disordered autonomic response to stimuli below the level of the spinal cord lesion. In an awake patient, this can present with headache, flushing, nasal congestion, sweating and piloerection. The most dangerous effect is hypertension, which may be severe and could lead to intracranial haemorrhage, myocardial ischaemia and arrhythmias. It is more common with high spinal lesions – particularly those above T6 – and seems to be caused by loss of descending inhibition and altered neuronal connections within the distal spinal cord. Compensatory mechanisms are intact above the level of the lesion, leading to vasodilation and flushing above this level.

Common triggers arise from caudal root levels, in particular bladder distension. The patient may already be receiving prophylactic agents such as clonidine to try and minimise cardiovascular instability.

This is a difficult question. The pathophysiology of dysreflexia is tricky. It should be enough that you are aware of it.

Why might lesions above T6 be of particular concern?

Lesions above this level involve the splanchnic circulation leading to an increased severity of symptoms.

Please go on with your anaesthetic management . . .

You can use this to demonstrate your understanding of the long-term multisystem effects of spinal cord injury.

Ideally this patient would need thorough preoperative assessment in a dedicated multidisciplinary preoperative assessment clinic prior to his scheduled procedure.

Preoperatively I would review the patient's notes, looking for details surrounding the original admission with the injury and any subsequent anaesthetics. In particular I would like to know the exact level of the injury, any airway difficulties, whether the patient required a tracheostomy at the time of his injury, ventilatory difficulties, including the need for postoperative and long-term non-invasive or invasive ventilation, and any previous history of autonomic dysreflexia.

I would take a full anaesthetic history and examine the patient, again focussing on these areas. I would structure this by body system to identify any issues related to his cord injury.

I would perform a thorough airway assessment as previous spinal cord fixation might limit his neck movement leading to difficult airway management. I would enquire about symptoms of gastro-oesophageal reflux as most spinal cord-injured patients have delayed gastric emptying, which may increase the risk of regurgitation and pulmonary aspiration under general anaesthesia. I would therefore consider prescribing antacid and prokinetic therapy. Given the level of his lesion, he may also be dependent on some form of ventilatory support, so I would specifically enquire about this. I would also ask about symptoms of obstructive sleep apnoea, which is more prevalent in this patient group, and then complete a STOP-BANG questionnaire.

You have mentioned he had a complete transection of the cord at C5, so I would expect him to have no sensation or motor power below that level. I would clarify this with him and assess any requirement for analgesia, taking into account any co-existent chronic or neuropathic pain resulting from his cord injury.

Contractures may lead to difficulties with positioning, vascular access, or surgical access. In addition, I would ask him about muscular spasm, which could interfere with surgery. I would also enquire about any history of cardiovascular disease, which again is more common in this patient group.

Following this, I would obtain baseline vital signs and arrange relevant preoperative investigations based on my findings from the patient's history, examination and clinical notes. This might include blood tests, routine microbiological screening and further objective assessment of his respiratory and cardiac function.

With regards to the anaesthetic plan, after clarifying positioning and estimate of duration with the surgeon, I would discuss with the patient the anaesthetic options. These include no anaesthesia (with or without sedation), central neuraxial blockade and general anaesthesia. In all cases, an anaesthetist needs to be present or available, in case the patient develops autonomic dysreflexia. If the patient was in agreement and there were no prior symptoms or signs of autonomic dysreflexia, and he had no sensation, then the simplest approach would be for him to remain awake for the procedure.

And what if there was a history of dysreflexia?

If there was a history, or if the risk was unknown, I would suggest central neuraxial blockade with a spinal anaesthetic to abolish the risk.

Your patient declines central neuraxial blockade and asks to 'have a general anaesthetic', how would you proceed?

In that case, I would first seek to clarify his concerns and any misconceptions about spinal anaesthesia, for example if he fears worsening of his lesion. Or whether he simply wishes to be 'asleep' and light sedation might be sufficient. If he still insisted on general anaesthesia, I would seek senior anaesthetic advice and explain the increased risk of general anaesthesia including difficult airway management, risk of aspiration and potential need for postoperative ventilatory support. If the consensus was that a general anaesthetic was reasonable, I would go ahead and prepare for that. Given his C5 lesion, unless there was good evidence of previous uneventful operations, without any respiratory complications, I would request a high dependency bed for his postoperative care.

And how would you anaesthetise him?

Again here you should have a well-rehearsed structured approach for describing delivery of anaesthesia for a given case.

I will consider induction, maintenance, monitoring, position and emergence.

Induction first. With trained assistance and all my usual checks, I would establish large bore intravenous access, which may be difficult with atrophic skin and poor blood flow. As such, I would have a low threshold for an ultrasound-guided approach to minimise the number of attempts.

The patient may have a reduced blood volume, so I would consider a crystalloid fluid preload. I would also have prepared an α -agonist such as metaraminol, anticipating exaggerated hypotension on induction of anaesthesia. As with any induction, I ensure atropine is immediately available.

If there were any concerns regarding his airway, I would consider an awake fiberoptic intubation. Otherwise I would carry out a modified rapid sequence induction with propofol and 1.2 mg/kg rocuronium as a rapid onset non-depolarising alternative to suxamethonium. I would be mindful that these patients have altered pharmacokinetics due to reduced muscle mass and blood volume, and hence may require lower doses of induction agent.

Why avoid suxamethonium?

In spinal cord injury there is a proliferation of acetylcholine receptors throughout the muscle, away from the motor endplate, presumably as a response to the sudden loss of efferent transmission. The use of suxamethonium can therefore lead to a massive depolarisation with subsequent hyperkalaemia which may lead to life-threatening arrhythmias and cardiac arrest.

Do you know how long the risk lasts?

I have read various estimates of the time period. Taking a cautious approach, I would avoid suxamethonium from 48 hours after the initial event, until 9 months later.

In all circumstances?

Every case would have to be assessed on a benefit/harm basis. The potential for a hyperkalaemic crisis within that time period is unquantifiable. Hyperkalaemia is also treatable with calcium, insulin and other measures. It is possible to imagine a situation where an airway crisis may lead to the need to use suxamethonium despite the risk. In an elective case, such as this, it should be avoidable.

These types of questions are more about awareness of the issues and a safety first approach, than any one technique.

You successfully intubate the patient and after a period of initial hypotension, the patient remains stable.

You've described induction. Please continue.

So on to maintenance. I would ensure adequate depth of anaesthesia using total intravenous anaesthesia with propofol and processed EEG monitoring. I would also use remifentanyl to minimise the risk of muscular spasm or dysreflexia. I would use intermittent positive pressure ventilation to avoid the increased risk of hypoxia or hypercapnia with his high cord lesion, while being alert for the exaggerated hypotension that may ensue.

The original question was 'how would you anaesthetise'. You don't have to go through all possible methods, just what you would do. Not everyone would use TIVA for example.

I would actively warm the patient, using a forced-air warmer and fluid warming, as he is likely to have altered thermoregulation. Although I would not anticipate major blood loss, I would frequently monitor the surgical site and replace any losses

immediately as the patient will not have the normal cardiovascular compensatory mechanisms. I would also have a rapid-acting antihypertensive such as glycerine trinitrate immediately available for any episodes of dysreflexia. As with any patient, I would take care with positioning using padding to protect sites vulnerable to pressure damage, which is a particular risk as his skin is likely to be thin and relatively avascular. As well as standard monitoring as per Association of Anaesthetists' guidelines, I would consider using intra-arterial blood pressure monitoring.

Would you use it or not?

As I do not have experience with these patients, and given the concerns with both hypo- and hypertensive crises, I would use it.

In fact some centres with a large volume of surgery for these patients, don't routinely employ intra-arterial monitoring. Given you have already explained the risk of cardiovascular instability it is probably easier to justify its use than not.

Despite all your preventative measures, during surgery his blood pressure rapidly rises to 210/140 mmHg. What would you do?

This is almost certainly autonomic dysreflexia. I would immediately send for senior assistance, ensure the patient is adequately anaesthetised, inform the surgeons and ask them to stop. I would sit the patient head up and ensure that any tracheal tube ties are loosened around the neck. If withdrawing surgical stimulus did not reduce blood pressure, I would increase the depth of anaesthesia. If that failed to obviate the hypertensive crisis, I would administer intravenous glycerine trinitrate if available, or via metered dose oral spray. Bladder distension would be a likely cause and I would ask the surgeons to urgently decompress it.

Intravenous GTN is faster acting and can be titrated to response. In high volume centres where central neuraxial blockade is routinely used to obviate dysreflexia, hypertensive crises are rarely encountered. A metered dose spray may be kept immediately available in theatre.

Why use glycerine trinitrate?

The hypertension is likely to be paroxysmal and therefore any agent must be rapid-acting and have a short duration of action. An alternative is the α -blocking agent phentolamine.

There is a large list of drugs in the literature that have been used to prevent or treat sympathetic dysreflexia including reserpine, doxazosin, nifedipine, hydralazine, magnesium and clonidine (which treats spasm as well as hypertension). Knowing one or two should suffice.

Having treated that crisis, the patient once again stabilises. You are nearing the end of the case and the surgeon informs you he plans to infiltrate with some bupivacaine and adrenaline.

What do you think about this?

I would suggest he does not use it. I would explain that the patient has a complete lesion, and therefore will not require the analgesia. If there was some concern about sensation then I would insist that he use plain local anaesthetic. The patient has already had one hypertensive crisis and is likely to have increased sensitivity to catecholamines.

Catecholamine levels, especially levels of noradrenaline, increase during dysreflexic episodes. Despite this they remain lower than levels in normal, non-injured patients. This implies that an increased sensitivity is at least partly responsible for the crises.

Most anaesthetists should be able to answer questions about the acute management of the patient with a spinal cord injury – you would be expected to be confident and decisive in your answers. However, the management of late complications from spinal cord injury deals with a specific patient group many anaesthetists will never encounter. Don't worry if you don't know all of the details, especially the pathophysiology of autonomic dysreflexia. Most of the issues you can work out – for example the risk of respiratory compromise. An awareness of the issues surrounding suxamethonium and hypertensive crises should be sufficient.

Further Reading

Petsas A, Drake J. Perioperative management for patients with a chronic spinal cord injury. *BJA Education*. 2015;15(3):123–130.

Rizk A, Saad M. et al. Perioperative complications and anesthesia practices in managing patients with quadriplegia undergoing surgery: A systematic review. *Frontiers in Medicine*. 2022;9.

2.5.6 Brainstem Death Testing – Benjamin Hofland-Ward and Susanna T Walker

This is a question that I was actually asked in my Final FRCA structured oral examination. It was asked as part of the anatomy structured oral examination. Guidelines have been adapted from the Academy of Medical Royal Colleges Code of Practice published in 2008.

What is brainstem death?

Brainstem death was defined formally at the conference of the Royal Colleges in 1976, and it is this definition that has since been used in the English law courts. It is defined as 'irreversible loss of the capacity for consciousness, combined with irreversible loss of the capacity to breathe'.

Please could you talk me through the functions of all the cranial nerves and tell me which ones are tested when we perform brainstem death testing?

The first cranial nerve (CN I) is the 'olfactory nerve'. This gives us the sense of smell, which is not tested during brainstem testing.

The second cranial nerve (CN II) is the 'optic nerve'. This nerve innervates the eye and enables us to see. It is involved as the afferent limb of the pupillary light reflex. This nerve is tested in brainstem testing by shining a bright light directly into the eye. A normal response would lead to constriction of the pupil. If someone were brainstem dead, there would be no response to light.

The third cranial nerve (CN III) is the 'oculomotor nerve'. This nerve innervates all of the muscles of the eye except the superior oblique and lateral rectus and is therefore involved in movement of the eye. This is not directly tested in brainstem testing. However, the third nerve is indirectly tested, as parasympathetic efferent fibres, which

control pupillary constriction, travel with the third nerve and are therefore tested as the efferent limb of the pupillary light reflex.

The fourth cranial nerve (CN IV) is the 'trochlear nerve'. This innervates the superior oblique muscle of the eye enabling the eye to look medially and down. This is not directly tested during brainstem testing.

The fifth cranial nerve (CN V) is the 'trigeminal nerve'. This is a large nerve with sensory and motor components. It supplies sensation to the face and scalp including sensation to the cornea, and motor supply to the muscles of mastication. This therefore supplies an afferent limb for the corneal blink reflex. This is tested during brainstem testing by lightly touching a strand of cotton wool onto the cornea. A normal response would be to blink, which is absent if someone is brainstem dead.

The sixth cranial nerve (CN VI) is the 'abducens nerve'. This innervates the lateral rectus muscle of the eye enabling lateral movement of the eye. This is not directly tested during brainstem testing.

The seventh cranial nerve (CN VII) is the 'facial nerve'. This is predominantly a motor nerve supplying muscles of facial expression. This is the nerve that results in blinking, and is therefore the efferent limb of the corneal blink reflex, which is tested in conjunction with CN V.

The eighth cranial nerve (CN VIII) is the 'vestibulocochlear nerve'. This has two main components; the cochlear and vestibular nerves, which are responsible for hearing and balance respectively. Damage to the vestibular nerve therefore causes problems with equilibrium and balance leading to vertigo. Often the most obvious clinical sign of this is nystagmus of the eyes. This nerve is tested in brainstem testing as part of the vestibulo-ocular or caloric reflex. It is important to check that the auditory canal is free from earwax prior to testing as this would affect the results. To perform the test the ear canal is irrigated with 50 ml of ice-cold water. Normally this would lead to eye deviation towards the side being tested and a nystagmus with the fast phase going away from the side being tested. In a brainstem dead patient there is no response. The afferent nerve for this reflex is the vestibular nerve, and the efferent limb indirectly tests the third, fourth and sixth cranial nerves (eye movements).

The ninth cranial nerve (CN IX) is the 'glossopharyngeal nerve'. This is predominantly a sensory nerve and supplies sensation to the posterior third of the tongue, and the pharynx. This is tested during brainstem testing as it forms the afferent limb of the gag reflex. Patients who are brainstem dead have no afferent portion to the gag reflex and therefore do not gag when a spatula is placed at the posterior pharyngeal wall.

The tenth cranial nerve (CN X) is the 'vagus nerve'. This is a large nerve supplying parasympathetic motor innervation to many organs in the body, and importantly motor innervation to most of the pharynx and larynx via the recurrent laryngeal and superior laryngeal nerves. This is tested during brainstem testing in two ways. Firstly, by placing a suction catheter down the endotracheal tube, normally this would elicit coughing, but there is no response in a brainstem dead patient. The vagus nerve is also involved in the gag test as it forms the efferent limb of the gag reflex.

The eleventh cranial nerve (CN XI) is the 'accessory nerve'. This innervates the sternocleidomastoid and trapezius muscles, and is not tested during brainstem testing.

The twelfth cranial nerve (CN XII) is the 'hypoglossal nerve'. This is the motor supply to the tongue and is not tested during brainstem testing.

Cranial nerves I, XI and XII are the only ones that are not tested in any way, as they do not contribute to a brainstem reflex arc.

What preconditions are required before performing brainstem death testing?

There are certain preconditions that should be met before brainstem testing is performed. Firstly, the patient should have an irrecoverable condition causing brain damage, which can lead to brainstem death. Conditions could include a severe head injury or a subarachnoid haemorrhage for example. The patient should also be in an apnoeic coma, requiring ventilation.

Do you know of any factors that might exclude patients from brainstem death testing?

There are several factors that might mean that it is inappropriate to perform brainstem death testing. These are all factors that could themselves lead to a change in cerebral function affecting the Glasgow Coma Score of a patient, and therefore could potentially lead to inaccurate testing suggesting a patient is brainstem dead when they are not.

To perform the tests, the patient should not be hypothermic. The latest Academy of Medical Royal Colleges guidelines recommend that the core temperature should be greater than 34°C. There should be no metabolic or endocrine disturbance, such as hypoglycaemia or hypothyroidism that could lead to a coma. However, it is well known that brainstem death itself may lead to metabolic and endocrine disturbances such as diabetes insipidus causing hypernatraemia. These may be as a result of rather than the cause of lack of brainstem function, and do not preclude brainstem death testing. There should be no evidence of depressant drugs causing the coma. Any sedation that may have been given on intensive care should have been switched off for an appropriate length of time to be certain that there is no chance of any residual effects of sedative medications. If any neuromuscular blocking drugs have been used, then a nerve stimulator should be used to demonstrate that there is no residual effect of these drugs. Testing cannot be performed on patients with a medical cause of paralysis such as Guillain-Barre' syndrome.

How would you actually perform the tests?

When the relevant preconditions and exclusion criteria have been addressed, the tests can be performed. Two doctors should perform the tests; they should both have been registered for more than 5 years, one of them should be a consultant, and neither can be involved in the hospital's transplant team. A complete set of the tests should be performed twice with the doctors acting together. There is no stipulation about how far apart the two sets of tests are performed, other than that baseline parameters should be restored before starting the second set of tests.

I have already described the nerve roots that are tested. However, in practice the tests are performed by first testing for a pupillary light reflex in both eyes. Then test for the corneal reflex in both eyes. Next, check that both ear canals are clear and then test for the vestibulo-ocular reflexes bilaterally. Ensure that there is no facial movement in response to a central painful stimulus, such as supraorbital pressure. It is important to note that

spinal reflexes may persist, leading to movement of limbs. This may be distressing for relatives to see but does not affect the outcome of brainstem testing. Then confirm that there is no gag or cough reflex.

Finally, the apnoea test is performed. The patient should be preoxygenated with 100% oxygen for 10 minutes. This is often done while the earlier tests are performed. The ventilation rate should be reduced during this period to allow the end-tidal CO_2 to rise above 6.0 kPa prior to disconnecting the ventilator. An arterial blood gas sample should be performed at this stage to confirm the PaCO_2 is at least 6.0 kPa and that the pH is less than 7.4. The ventilator is then disconnected, and the patient is oxygenated by insufflating oxygen at 5 L/min into the lungs via a suction catheter placed down the endotracheal tube. If after 5 minutes, the patient has shown no signs of respiratory effort a further blood gas sample is analysed to ensure that the PaCO_2 has risen from the starting level by more than 0.5 kPa. The PaCO_2 usually rises by 0.4–0.8 kPa/min depending on the metabolic rate. Patients with pre-existing lung disease may normally have a raised PaCO_2 , therefore an appropriate starting PaCO_2 for this patient should be determined on the basis of a blood gas demonstrating a mild acidaemia with a pH less than 7.4. The patient should not be allowed to become hypoxic while these tests are being performed. CPAP may be used to maintain oxygenation if required. Occasionally apnoea testing may have to be abandoned as a result of oxygen desaturation.

What are ‘doll’s eye movements’? Are these required as part of brainstem death testing?

Doll’s eye movements describe the oculocephalic reflex. In an unconscious patient, if their head is turned to the side, the eyes rotate in the opposite direction, therefore keeping the eyes pointing in the original direction in relation to the surroundings – as is seen in a child’s doll. This reflex requires an intact brainstem, and therefore in a brainstem dead patient, when the head is turned, the eyes move in the same direction as the head. This is not a test that is required in brainstem testing in the UK, although may be tested in other countries.

What clinical features may be seen that are associated with brainstem death?

An injury to the brain leads to swelling of the brain within the skull, raising intracranial pressure which will lead to a reduction in cerebral perfusion and therefore a reduction in oxygen supply to the brain. All of the subsequent clinical features are related to this brain swelling. The final result of the swelling if this continues is for the brainstem to be forced through the foramen magnum. This is known as ‘coning’ and leads to brainstem death. There are various clinical signs that may be elicited at various stages as a result of the brain swelling. A third nerve lesion may be seen as a result of herniation of the uncus. A sixth nerve lesion may be seen as swelling stretches the nerve. The Cushing’s reflex occurs as a desperate attempt to improve cerebral perfusion. This leads to significant hypertension associated with a bradycardia, which is the result of the baroreceptor reflex. Various arrhythmias may then subsequently occur. There are also several endocrine manifestations of brainstem swelling, such as a failure of thyroid hormone synthesis due to hypothalamic and pituitary failure. This further exacerbates cardiovascular changes

and instability. There is also a lack of antidiuretic hormone, which results in neurogenic diabetes insipidus, leading to large quantities of very dilute urine. Finally, there is a loss in thermoregulation, which usually leads to hypothermia.

Do you know of any additional tests that may be required in other countries to confirm brainstem death?

If you're being asked these questions you're doing well – keep going!

In the UK there is no legal requirement to perform any tests in addition to brainstem testing. However, in certain situations these may be considered if, for example, pre-existing lung disease makes it impossible to complete the apnoea test as a result of oxygen desaturation, or if it is not possible to test some of the cranial nerves as a result of craniofacial trauma. In some countries, such as the United States, extra tests are commonly performed. The tests that may be performed aim to elicit a complete lack of electrical activity and blood flow to the brain. The gold standard to demonstrate lack of blood supply to the brain is four-vessel angiography. A radio-opaque contrast medium can be injected into both internal carotid and both vertebral arteries. In brainstem death flow is obstructed due to raised intracranial pressure. More recent, and less invasive, techniques include CT angiogram and MR angiogram, both of which also demonstrate the lack of blood flow. Transcranial Doppler ultrasonography is also mentioned, where a characteristic change in the velocity waveform of the basal cerebral arteries is seen. This is the least invasive test and can be performed at the bedside in ITU.

An EEG may be performed to demonstrate a lack of electrical activity. There are some controversies regarding the EEG as it is technically very difficult to perform in the ITU setting due to the large amount of interference from surrounding equipment. It is also possible to perform auditory evoked potentials (AEPs) to demonstrate a lack of brain activity.

What is the legal time of death?

The legal time of death is the time at which the first set of tests has been completed. However, the patient is not actually pronounced dead until after the second set of tests have been performed.

Why do we have a shortage of organ donors in the UK compared with the rest of Europe?

This is quite a topical subject that has been in the news a lot over the past few years.

The UK donation rate was one of the lowest in Europe with a study from 2006 showing only 12 per million of the population were prepared to donate their organs for transplant, compared with 33 per million of the population in Spain. The rate of donation had actually fallen in the UK. It is not known why this was, but it was thought that one of the main reasons was that relatives and family members were reluctant to allow transplantation procedures. During the 2-year study, by the UK Transplant organisation, of the potential organ donors whose families were approached for donation 41% of the families denied consent. The main reasons for refusal were that they did not want surgery on the body, they were not sure if the patient would have agreed, or that the relatives were divided. The refusal rates were highest amongst families from ethnic

minorities (70%) in comparison to those of white donors (35%). It should be noted that organ donation was only discussed in 94% of potential cases, meaning that 6% of cases were missed opportunities. Therefore, in a small part, the lack of organs is a result of clinicians not discussing donation with the next of kin. This is a difficult subject to broach, and therefore there may be some delay or reluctance on the part of the clinician. To address this issue there has been a drive to increase numbers of organ transplant coordinators. They provide an important service as they are specifically trained to broach this issue and are often based within the hospital and can therefore be called upon very quickly to talk to relatives and initiate the required procedures leading to organ donation. There is also the benefit that they can identify inappropriate patients quickly and therefore prevent an unnecessary discussion with the family, which avoids causing further distress. They also have an important role of increasing awareness of organ donation amongst the general population.

The shortage of organs can also be attributed to the state of brainstem death being uncommon in the UK. This is partly due to a decrease in deaths from road traffic accidents, improved trauma care and a decrease in cerebrovascular deaths.

How may this shortage in donor organs be addressed?

A strategy set out in June 2021 set the ambition for the UK to be a world leader in organ donation and transplantation. As a result of approaches described above, the rate of donation has increased in recent years with the consent/authorisation rate of eligible patients after circulatory death rising from 51% in 2013 to 65% in 2019/20. In addition, the most significant event in May 2020 was a change in the UK organ donation law to an 'opt out' system. This means anyone is presumed to be an organ donor unless they have specifically opted out. Family members are, however, still involved in the decision-making process. It is hoped that over the next 5 years this change will lead to an increase in consent/authorisation to an average of 80%.

A specific focus on Black, Asian and Ethnic minority groups forms part of the new strategy, by working with charities and local communities. We know that families from these groups are less likely to discuss organ donation, are less likely to want to donate an organ, but more likely to need a transplant than the rest of the UK population.

What criteria would suggest to you a brainstem dead patient may not be suitable as an organ donor?

The main criteria that can be applied to organ donation are firstly to prevent transmitting a transmittable disease such as infection or malignancy and secondly, to ensure acceptable function of the donor organ. Various absolute and relative contraindications to transplant have been suggested.

Absolute contraindications would include age over 80 years, active metastatic cancer, DIC and sickle cell anaemia. HIV infection is an absolute contraindication, although in some states in America, transplants between patients infected with HIV have been allowed in certain circumstances. Prolonged hypotension or hypothermia is also an absolute contraindication, although a brief period of resuscitated cardiac arrest is considered acceptable when putting a patient forward for organ transplant. Ultimately, the transplant team makes the final decision – often in theatre – regarding the suitability of organs.

Relative contraindications would include malignancy that has been in remission for over 5 years, hypertension, diabetes mellitus, physiological age greater than 70, hepatitis B or C infection, and a history of smoking. Some heart, lung, liver and renal dysfunction may be acceptable if it was caused in the context of trauma leading to brainstem death. Brain tumours are not an absolute contraindication, although it must be certain that the brain tumour does not represent a metastasis from another primary source.

Further Reading

Academy of Medical Royal Colleges. A Code of Practice for the Diagnosis and Confirmation of Death. Academy of Medical Royal Colleges. Published October 2008. www.aomrc.org.uk/publications/reports-guidance.html.

Barber K, Falvey S, Hamilton C, Collett D, Rudge C. Potential for organ donation in

the United Kingdom audit of intensive care records. *British Medical Journal*. 2006; 332 (7550): 1124–1128.

Organ Donation and Transplantation 2030: Meeting the Need. A ten-year vision for organ donation and transplantation in the United Kingdom. UK Health Ministers. Launched 1 June 2021.

2.5.7 Anaesthesia during an MRI Scan – Farzad Saadat and Sarah F Bell

This topic might be part of a short clinical or a case with a linked science question. An extensive, in-depth understanding of the workings of the MRI is not required. The examiner is looking to see whether you have a basic grasp of the physical concepts and whether you are aware of the safety issues that exist.

Can you start by telling me how an MRI scanner works?

Try and avoid getting bogged down with complicated physical principles.

The MRI machine produces an extremely strong magnetic field. This causes the protons within the hydrogen atoms of water to become aligned, either in the same direction as the magnetic field or in the opposite direction. The high water content of the body is vital to the formation of a magnetic resonance image.

A pulse of electromagnetic radiation (in the form of radio waves) causes the hydrogen nuclei to rotate from their equilibrium position. When this pulse is then removed the nuclei emit radio waves as they return to their original equilibrium position. This radio wave signal is detected by the coil in the MRI and processed by the computer system to produce a visual display.

What are the components of an MRI scanner?

The MRI machine is composed of a primary magnet, gradient magnets, a coil and a computer system to analyse the data.

Can you tell me more about the primary magnet?

Magnetic fields are produced by ferromagnetic materials such as iron, cobalt or nickel. A magnetic field may also be generated by passing a current through a wire. The primary

magnet in an MRI scanner consists of a coil of wire through which a current is passed. The wire is made from a superconducting material. This material has no electrical resistance below a certain temperature. It is therefore able to produce a very large magnetic field. The low temperature required is in the region of minus 260 degrees Celsius (or just above absolute zero). To achieve this temperature the wire is kept in liquid nitrogen and helium.

What can you tell me about the gradient magnets and the coil?

There are three smaller magnets within an MRI machine called gradient magnets. These magnets are much weaker than the primary magnet. They allow precise alterations of the magnetic field. They are important in generating the final images.

The coil is the part of the MRI machine that emits and receives the electromagnetic radiation, in the form of radio waves.

How strong is the magnetic field produced?

The MRI scanner produces a magnetic field of 1.5 to 3 Tesla.

What does Tesla mean?

Tesla is the unit of magnetic flux density. This is a measure of the strength of a magnetic field. Another unit used is the Gauss. Ten thousand Gauss is equal to one Tesla. The earth's magnetic field is equal to between 0.5 and 1 Gauss.

What is the 50 Gauss line?

The magnetic field of 50 Gauss is marked around an MRI scanner with a line. This is the 50 Gauss line.

The images produced are called either T1 or T2 weighted images.

What does this mean?

T1 and T2 are called the relaxation time constants. They are different measures of how the hydrogen nuclei relax back to their original alignment. Either T1 or T2 is used to provide extra contrast to an image. In T1 fluid is seen as dark, whereas in T2 fluid is white. This can be useful in deciding the type of tissue seen in an image.

You are asked to transfer a 57-year-old ICU patient to the MRI suite. They require further investigation, having not woken from sedation hold following an out of hospital cardiac arrest 5 days previously. They are intubated, but otherwise stable and not requiring organ support. What are the challenges of taking this patient for an MRI scan?

This question is testing your ability to recognise the many problems that might occur. Try and give some broad categories before going into detail.

This is a complex scenario, which can present problems in a variety of areas. These can be divided into patient-associated problems, problems with transfer of an intubated

patient, problems with administering an anaesthetic in a remote and unfamiliar site and MRI specific problems. The MRI problems can be subdivided into monitoring and equipment issues and safety issues.

Can you tell me anything about the patient-associated problems?

Yes. Any transfer of an intubated ICU patient is complex and requires a full ABCDE assessment and plan. The grade of difficulty of the patient's intubation must be checked, as tubes can become dislodged in transfer, or in MRI. The tube should be secured in such a way to minimise this risk. If the patient is breathing spontaneously, paralysis should be strongly considered, to minimise movement in the MRI or problems with ventilation. ICU patients often have multiorgan support requirements. Every pump delivering any vital drugs, such as vasopressors or blood pressure management should be fully charged, with back up pumps and drugs. Any non-vital infusions (such as feed) can be temporarily stopped. The patient will require continuous monitoring throughout.

ICU infusion pumps and monitoring equipment are not usually MRI safe, and so safe transfer onto MRI monitoring needs to be considered.

Can you talk me through the monitoring and equipment problems?

The monitoring problems may be split into dangers posed by non-MRI compatible equipment, difficulties obtaining a reading and then complications with interpretation of a value.

The 50 Gauss line is marked on the floor of the MRI. This is an important boundary. Ferromagnetic items such as iron or molybdenum steel that are within this line will move and potentially act as projectile objects. There are also other materials that can behave differently and unpredictably in a magnetic field. All monitoring devices need to be able to function reliably under these conditions. Changing to MRI compatible equipment should take place before entering the scanner.

All patients who require an anaesthetic should be monitored as per the AAGBI minimum monitoring standards. The MRI tube is narrow and it can be difficult to access the patient during the scan. The monitoring therefore needs to be secure. The scan may take a variable length of time which can lead to hypothermia in some patients. It is therefore important to consider warming devices and the ambient room temperature in certain cases.

The ECG electrodes are made of non-magnetic materials. The cables connecting the pulse oximeter and ECG are fiberoptic. Care should be taken to avoid looping of cables and to pad the skin underneath them. This is to reduce the chances of burns to the patient. Burns might occur if currents are induced in the cables causing an increase in their temperature.

Can you tell me about the problems with interpreting the monitoring?

With regards to interpreting the monitoring, MRI can have some important effects.

Firstly, let's consider the ECG. The magnetic field can generate currents in the blood as it flows through the thoracic aorta. This can result in artefact in the ST region of the ECG, or a reduction or even inversion of the R-wave. Monitoring for myocardial

ischaemia can therefore be unreliable, but the ECG should revert to normal once scanning stops.

Secondly, the capnography and gas analysis are affected. This is because of the length of sampling tubing required to bring the gases out of the MRI scanner to the analysing equipment. Delays of up to 20 seconds can occur.

Finally, the MRI is extremely noisy. Acoustic protection is required and so audible alarms are useless. The monitoring therefore needs to have visible alarms. The anaesthetist needs to ensure that they have an unobstructed view of the screen at all times.

What is the difference between MRI safe and MRI compatible equipment?

‘MRI safe’ means that there will be no safety issues when the equipment is taken into the MRI. It does not guarantee that the equipment will function normally or not interfere with the correct operation of the MRI. ‘MRI compatible’ equipment is MRI safe and does not interfere with the operation of the MRI.

Is the anaesthetic machine used for MRI safe or compatible?

The anaesthetic machine used in the MRI room must be MRI compatible. This includes the vaporisers and the gas cylinders.

What about your infusion pumps?

These should be MRI compatible. Some pumps will fail if the field strength is greater than 100 Gauss.

What type of circuit do we commonly use in the MRI scanner?

A Bain circuit is frequently used in an MRI scanner. It can be up to 5.4 m in length while maintaining low deadspace and resistance.

How would you minimise the hazards posed to this patient when in MRI?

Try and structure your answer so that the examiner is aware that you know there are many possible complications.

The hazards may be due to the magnetic field, the noise, the anaesthetic gases and the potential for MRI malfunction. Magnetic field hazards may be further divided into projectile effects and burns.

The patient should be screened for implanted ferromagnetic objects, this includes pacemakers, aneurysm clips and metal foreign bodies in the eye. The patient’s records should be searched, as well as collateral history from any relatives and a physical examination of the patient for any such hazards.

The MRI can cause burns by inducing currents in the monitoring cables, which will then heat up. Care should be taken to avoid coiling of wires, and pads should be placed between them and the patient’s skin.

The MRI produces a large amount of acoustic noise and so protection should be worn. This may be in the form of ear plugs or defenders.

During administration of an anaesthetic there is the risk of anaesthetic gas inhalation if the scavenging equipment is not as effective as that in theatre. There are MRI compatible systems produced that should be used whenever possible.

If the MRI malfunctions or needs to be shut down, there is a risk of hypoxia.

Why is this?

When the MRI is shut down the liquid helium and nitrogen that surrounds and cools the superconductor is allowed to rapidly evaporate and escape from inside the magnet. If there is inadequate ventilation of the area, a fall in the oxygen concentration will occur. This can lead to a hypoxic environment.

Are there risks to staff?

Many of the same patient risks apply to staff, who must also be screened for ferromagnetic objects. The staff and patient also need to remove any ferromagnetic jewellery or ID badges, credit cards etc.

The current maximum safe level for exposure to the intense magnetic field is 200 mT within an 8-hour period.

There are potential hazards in pregnancy, including risk of hyperthermia and teratogenicity although no proven adverse effects. Pregnant staff are advised not to remain in the scan room while imaging is underway, primarily due to concerns about acoustic noise exposure.

Given all the risks of the MRI, why do we not just use a CT scan for all cases that require imaging?

The CT is also not without risk. The same challenges of remote site anaesthesia, patient- and procedure-related factors still occur. In addition, the patient is being exposed to X-ray radiation that can predispose to malignancy. The radiation exposure for a CT chest is approximately 50 times that of a plain X-ray.

The images produced by CT and MRI are different. The techniques are therefore used for to view and diagnose different conditions. MRI is particularly good at imaging the nervous system and pelvic disease. CT is useful for imaging bony deformities.

Do both techniques use contrast medium?

Yes, in both techniques the radiologist may require contrast medium to be given. This can cause anaphylaxis.

The MRI contrast most commonly used is gadolinium DTPA which can cause nausea, vomiting and pain on injection.

The CT contrast mediums often contain iodine. This can cause acute renal failure in susceptible patients. The patient should be well hydrated prior to administration of the medium. If the patient has renal impairment or diabetes then the contrast medium may be avoided.

Are you aware of any guidelines regarding anaesthetising patients in the radiology suite?

The Association of Anaesthetists have produced online guidelines regarding the provision of anaesthetic services in the MRI scanner.

Further Reading

Wilson SR, Shinde S, Appleby I et al.
Guidelines for the safe provision of
anaesthesia in magnetic resonance units.
Association of Anaesthetists. 2019.

Igra MS, Skipper NT, Davidson A.
Radiological investigations in
neuroanaesthesia and neurocritical care,
part 2: Magnetic resonance imaging *BJA
Education*. 2018.

2.5.8 Depth of Anaesthesia Monitoring – Benjamin Hofland-Ward and Susanna T Walker

This is an important topic that you will be expected to know about.

What is awareness?

There are a few definitions that you should be able to recite to convince the examiners that you have an understanding of this subject.

Anaesthetic awareness is a serious complication, and one of the most feared complications, of general anaesthesia. Awareness can be classified into explicit memory, implicit memory and deliberate awareness.

Explicit memory can be defined as the intentional or conscious recollection of experiences. Anaesthetic awareness specifically refers to explicit memory of intraoperative events, which involves spontaneous or conscious recall. The explicit recall may occur with or without the sensation of pain and recollections may be vivid, such as operating room conversation, or vague, such as dreams, or unpleasant sensations associated with the operation.

Implicit memory is the perception of ‘something’ without spontaneous recall of events. The patient initially denies remembering anything but may subsequently remember ‘something’ under hypnosis or with repeated questioning.

Deliberate awareness occurs when patients are awake for surgery performed using local or regional anaesthesia. It also occurs during some neurosurgical procedures when the patient is woken up to assess whether surgery has affected, or will affect, important areas. This is, however, less common since the introduction of devices to monitor evoked potentials intraoperatively.

Can you tell me the incidence of awareness?

The incidence of awareness varies significantly depending on the clinical situation and the type of anaesthetic given. Overall, the incidence of awareness with explicit recall of severe pain is commonly quoted as being approximately 1 in 3000 (0.03%). The incidence of awareness without recall of pain is commoner, and overall, between 1 and 2 per 1000 people are thought to experience some sort of awareness. However, it is important to note that some of these figures may be exaggerated by memories generated during awakening or in recovery. A large survey published in 2007 found an overall rate of awareness of 1 in 14,000 general anaesthetics, and the more recent NAP5 study from 2014 found an overall rate of 1 in 19,000 general anaesthetics. This can be broken down into:

- 1 in 42,000 for patients with no risk factors
- 1 in 100 for cardiac surgery (although quoted as approximately 1:8,600 from NAP5)

- 1 in 20 for trauma surgery
- 1 in 250 for emergency caesarean section under general anaesthesia (approximately 1:670 from NAP5).

Awareness is almost twice as likely when neuromuscular blockade is used.

What are the consequences of awareness for the patient?

Awareness may have serious psychological repercussions for the patient, including insomnia, depression, post-traumatic stress disorder and a fear of future surgery.

What are the causes and risk factors for awareness?

Try to classify the causes and risk factors into a way that you will easily remember. There are many ways to do this. One might be patient factors, anaesthetic factors and surgical factors.

Patient factors:

- Disease processes (e.g., sepsis, hyperthyroidism)
- Social factors (e.g., alcoholism, recreational drugs)
- Medications (e.g., beta-blockers)
- Previous history of awareness.

Anaesthetic factors:

- Equipment malfunction
- TIVA technique
- Difficult intubation
- Use of neuromuscular blockade.

Surgical factors:

- Trauma surgery
- Cardiac surgery
- GA caesarean section.

Ultimately, awareness results from an inadequate anaesthetic dose. Light anaesthesia, especially when a patient has received a muscle relaxant, is associated with the highest risk of awareness. There are four main reasons why an anaesthetic may be too light.

Firstly, not giving a large enough dose of anaesthetic. This may occur when a volatile agent is accidentally not given or started late; or when an anaesthetic dose is reduced in a hypotensive patient to preserve the blood pressure. It is important to remember when selecting a dose that patients vary in their requirements, and often the chosen dose is based on patient averages. Minimum alveolar concentration (MAC), for example, is a population average and varies with age, gender and race.

Secondly, some patients, such as those with hypermetabolic states (e.g. pyrexia or hyperthyroidism), may have an element of resistance to anaesthetic agents.

Thirdly, there may be a problem with the equipment used. Breathing circuits may become disconnected, vapourisers may become empty, and TIVA infusion pumps may fail. For these reasons, a pressure fail alarm should always be used when ventilating patients. Volatile agent monitors with high and low alarm settings are widely available for use.

Finally, clinical signs of awareness, which would normally lead the anaesthetist to increase the anaesthetic dose, may be hidden. For example, patients taking beta-blockers are less capable of mounting a tachycardia.

When discussing causes and risk factors for awareness, it is important to mention the type of surgery and the type of anaesthetic being given.

Certain operations carry a much higher risk of awareness. These are generally situations where there is a risk of patient instability, so therefore an insufficient dose of anaesthetic agent may be given. These would include emergency GA caesarean section, high-risk cardiac surgery and acute trauma with hypovolaemia, where the incidence of awareness is quoted as being up to 5% depending on the severity of the trauma.

Patients with airway issues, including unexpected difficult intubation, or those undergoing rigid bronchoscopy are also at higher risk of awareness due to a failure to give any further anaesthetic agent during the period where it is difficult to intubate or ventilate the patient.

Finally, it is particularly important to note that the NAP5 study comments on the striking use of neuromuscular blockade and the incidence of awareness. While less than 50% of GA cases in the UK include the use of neuromuscular blockade, 93% of cases reported to NAP5 involved neuromuscular blocking agents.

What methods are you aware of for monitoring the depth of anaesthesia?

Think of a way to classify these as there are many, and you will then be less likely to forget the main important ones.

These could be classified into:

- Simple techniques
- Techniques which use a form of EEG monitoring
- Other techniques.

Simple techniques include monitoring for clinical signs of tachycardia, hypertension, tachypnoea, sweating and lacrimation. End-tidal agent monitoring is a simple monitor that is routinely used in most anaesthetics given in the UK. While this does not actually measure depth of anaesthesia it is a useful adjunct to ensure attainment of the desired concentration of anaesthetic agent. It does, however, have its limitations and should therefore only be used as a guide in conjunction with other clinical signs.

Techniques that use a form of EEG monitoring include pure EEG analysis, cerebral function monitors, frequency domain analysis, compressed spectral array, bispectral analysis and entropy. EEG monitoring uses 19 electrodes to create a trace of cerebral activity. It is therefore time-consuming, impractical to use during a standard anaesthetic, and complex to interpret, all of which limits its usefulness for monitoring the depth of anaesthesia. Different anaesthetic agents affect the EEG pattern in different ways and factors such as hypoxia, hypercarbia and hypotension can also lead to changes in the EEG. This makes interpretation of the EEG complex and impractical for use as routine monitoring. Cerebral function monitors, frequency domain analysis, and compressed spectral array are all monitors which process and modify the conventional EEG to simplify the information. Fourier analysis is used to process the raw EEG data into their component sine waves. These are then further analysed with respect to the frequency

distribution, the power contained within a waveform, that is, its amplitude, and the relationships between waves of different frequencies. The output is displayed in different ways, but overall leads to a trace that is easier to interpret than the raw EEG. None of these monitors are widely available for depth of anaesthesia monitoring.

Other techniques include the isolated forearm technique, lower oesophageal contractility, somatosensory evoked potentials and frontalis scalp EMG.

How useful is the monitor for lower oesophageal contractility?

There are two types of smooth muscle contraction that can be detected in the lower oesophagus – spontaneous contractions and provoked contractions. Both have a reduction in latency and amplitude with increasing depth of anaesthesia. The provoked contractions result from a sudden distension of the oesophagus, which can be achieved by rapidly inflating a balloon catheter placed in the lower oesophagus. A distal pressure transducer detects the elicited contraction. The evidence for using this as a depth of anaesthesia monitor is fairly limited and it is generally thought to not be reliable.

What do you know about the isolated forearm technique?

This is small-print information, so don't worry if you are being asked this!

This is a technique which is mainly historical and of interest for research. A tourniquet on the patient's upper arm is inflated above systolic blood pressure prior to giving muscle relaxants. Therefore, the arm with the tourniquet applied theoretically receives no muscle relaxant. Prior to induction of anaesthesia the patient is informed to move their arm if they are awake. After 15–20 minutes the tourniquet has to be let down to prevent limb ischaemia and subsequent temporary limb paralysis. It can be re-inflated if further relaxant is required. Research has shown that movement of the arm may not necessarily indicate explicit awareness, as the patient may have no recollection of moving their arm intraoperatively. It has also been argued that a response to command, or spontaneous movement, during surgery is a late sign when attempting to prevent awareness, and therefore this technique is not very helpful as a method of monitoring depth of anaesthesia.

How useful are evoked potentials at estimating depth of anaesthesia?

Of all the 'other techniques', this is the one to know something about. There is renewed research particularly into the use of auditory evoked potentials to monitor the depth of anaesthesia.

Evoked potential monitors measure the response, using a recording electrode, in specific areas of the brain when a supra-maximal sensory stimulation is applied to a peripheral nerve. An increase in latency and reduction in amplitude of these responses is seen with increasing depth of anaesthesia with most anaesthetic agents.

Visual evoked potentials are less reliable than auditory evoked potentials. When using auditory evoked potentials, a repetitive auditory stimulus at 6–10 hertz is played to the patient through headphones. A response waveform can then be plotted against time. Repeated stimulation enables the response signal to be identified against the background EEG. The waveform formed consists of three peaks. Firstly, the brainstem response; secondly the mid-cortical response; and thirdly the late cortical response. The brainstem

and late cortical responses are not affected by depth of anaesthesia. However, the mid-cortical response is that the amplitude of the waves is decreased, and the latency increased in a dose-dependent manner. By processing the raw waveform, the 'auditory evoked potential' (AEP) index can be calculated, giving a number which might be more meaningful to interpret than the raw waveform. A value of greater than 80 indicates an awake patient and a value less than 50 indicates an anaesthetised patient. There is a sudden increase in the AEP index when a patient changes from being unconscious to being awake. Therefore, it can be easier to distinguish between an asleep patient and an awake patient than with other monitors, such as the BIS. However, there is inter-patient variability for the index value at which this change takes place. Therefore, it is difficult to define a sensitive and specific cut-off value, while in an individual patient the pattern is very reproducible with awakening repeatedly occurring at the same index values.

What do you know about BIS monitoring? Are you aware of any recent studies into the use of BIS?

There are two or three reasonably important recent studies that you might be expected to have heard of even if you do not know the details well.

The BIS monitor obtains an EEG trace from fronto-temporal electrodes which is then processed into a number on a scale of 0–100; 100 represents normal brain activity, 0 represents complete absence of cerebral function, and a value of less than 60 is thought to represent a value below which there is a very low probability of postoperative recall. BIS values decrease with an increasing dose of anaesthetic agent, irrespective of whether this is inhalational or intravenous. The one exception to this is when using ketamine, which increases cerebral function, therefore increasing the BIS value. Studies have shown that BIS values decrease when muscle relaxant drugs are given, suggesting that the patient is at a deeper level of anaesthesia. However, the values do not change when opiate drugs or nitrous oxide are given, suggesting no change in depth of anaesthesia. These findings are obviously conflicting with what we know from clinical experience and should therefore make the anaesthetist cautious if delivering an anaesthetic to a targeted number.

There are several limitations to BIS monitoring. Firstly, there is no gold standard to compare BIS to, and so it is difficult to evaluate its effectiveness. The values themselves tend to have significant inter-patient variability. While one patient is asleep at a high BIS value such as 75, another may still be awake at a lower value of 70. The BIS values show a continuum from awake to asleep, and therefore there is no clear-cut point (unlike auditory evoked potentials) where transition is made from asleep to awake.

There are two major trials assessing the effectiveness of BIS monitoring. The first to mention is the 'B-Aware Trial' published in the *Lancet* in 2004. This was a large prospective, randomised, double-blind multicentre trial. The patients in the trial group were at high risk of awareness undergoing a variety of procedures including cardiac, trauma and obstetric surgery. They were randomly assigned to receive standard general anaesthesia, or a general anaesthetic with BIS monitoring. The patients were blinded to the protocol they received, as were the observers who assessed them for awareness at 2–6 hours, 24–36 hours and 30 days. The overall outcome from the study suggested that BIS-guided anaesthesia reduced the incidence of awareness with recall by approximately 82%. They also incidentally reported that the use of BIS

led to a reduction of administration of volatile gases. The group therefore concluded by recommending the use of BIS in high-risk patients. It was estimated that if we were to routinely use awareness monitoring for most patients in the United Kingdom it would cost approximately £30 million annually.

The other trial published was the 'B-Unaware Trial' which was published in the *New England Journal of Medicine* in 2008. This trial was based in Washington, USA. Once again, all of the patients included were considered to be at high risk of awareness under anaesthesia. There were various inclusion criteria, which were based on factors such as previous history of awareness, history of difficult intubation, ASA class 4 or 5, aortic stenosis, or end-stage lung disease. The patients were due to have a general anaesthetic with a volatile agent with or without nitrous oxide. The patients were randomly split into two groups. The first used BIS monitors and targeted values of 40–60. The second group used end-tidal agent monitoring targeting a MAC of 0.7–1.3. Alarms were set to alert the clinician when values were outside these ranges. Awareness was assessed using questionnaires – a well-accepted method – at 24 hours, 72 hours and 30 days. Patients and investigators were unaware of the groups to which they had been assigned. There were two definite cases of awareness in each group. The trial did not reproduce the results of the previous B-Aware Trial, and therefore did not show a reduction in the incidence of awareness with BIS monitoring. Also, the use of BIS did not result in a reduction in administration of volatile anaesthetic agents. Therefore, findings from this study do not specifically support the use of BIS as part of monitoring in standard practice.

More recent trends in the depth of anaesthesia monitoring include the use of technologies such as the *Conox* monitor. This includes the use of an index of nociception known as the qNOX. This scale ranging from 0 to 99 provides a probability prediction of response to noxious stimuli, with 99 being a highly likely probability of response. Therefore, it can be interpreted that the lower the qNOX value the lower the likelihood of response to a noxious stimulus and thus the greater the depth of anaesthesia. Research suggests this monitor may have less susceptibility to interference from electrocautery which, in the case of BIS, tends to lead to erroneously high values potentially leading the anaesthetist to give a higher level of anaesthesia than required.

Can you tell me what entropy is?

Entropy can be defined as being a quantitative measurement of disorder in a system. Entropy monitoring for depth of anaesthesia relies on assessing the degree of irregularity in the EEG signals. The amount of irregularity decreases with increasing brain levels of anaesthetic drugs. The monitor itself is similar to a BIS monitor in that it consists of EEG electrodes placed on the patient's forehead. The monitor processes the data collected and produces two different numbers. These are the response entropy (RE) and state entropy (SE). The SE reflects the cortical state of the patient while the RE incorporates EMG waveforms in addition to EEG waveforms, and is thought to measure adequacy of analgesia since painful stimulus may increase EMG activity. The RE looks at higher frequency waves and this enables a faster response time from the monitor. Manufacturers recommend targeting a range of 40–60 for both parameters by administering anaesthetic or analgesia accordingly. Studies have confirmed that entropy scores do relate to depth of anaesthesia.

How does entropy measurement compare with BIS?

Various studies have been performed trying to compare entropy and BIS in the measurement of depth of anaesthesia. Overall, it seems that they both perform similarly and that there is concordance between both and clinically observed depth of anaesthesia. It is notable that both BIS scores and entropy scores are unchanged by nitrous oxide use.

What should you be doing in your day-to-day practice to reduce the risk of awareness in your patients?

The Royal College of Anaesthetists published recommendations in January 2006. They stated that ‘close vigilance of all aspects of the patient and anaesthetic equipment’ together with ‘observations of changes to normal physiological variables’ should remain the principal factors in monitoring of awareness.

The NAP5 study in 2014 concluded with 64 recommendations. These included aspects relating to training such as ensuring all anaesthetists are familiar with depth of anaesthesia monitoring, and how to safely give a TIVA anaesthetic, and considerations such as avoiding NMB where possible, and always using a nerve stimulator when NMB has been given to ensure complete reversal.

In summary, we should strive to give a ‘good’ anaesthetic by firstly, checking all equipment thoroughly. Then, select an appropriate anaesthetic dose and identify those patients with increased requirements. Then, monitor the MAC or depth of anaesthesia of the anaesthetic agent throughout anaesthesia. When using MAC, we need to have an idea of what the MAC should be for that particular patient. It is important to remember that because MAC is a laboratory-derived average, it is only a guide and one should not strive to follow MAC numbers if clinical signs indicate otherwise. It has been suggested that a MAC of greater than 0.8 significantly reduces the risk of awareness. The risk of awareness can also be reduced by considering an awake regional technique, considering the use of benzodiazepines and avoiding neuromuscular blockade whenever possible.

Further Reading

Avidan MS, Zhang L, Burnside BA, et al. Anesthesia awareness and the bispectral index. *New England Journal of Medicine*. 2008; 358: 1097–1108.

Bein B. Entropy: Best practice and research. *Clinical Anaesthesiology*. 2006; 20(1): 101–109.

Guidance on the provision of anaesthesia services for intra-operative care 2009. Royal College of Anaesthetists. 2009.

Myles PS, Leslie K, McNeil J, Forbes A, Chan MT. Bispectral index monitoring to prevent awareness during anaesthesia: The B-Aware randomised controlled trial. *Lancet*. 2004; 363: 1757–1763.

Pollard RJ, Coyle JP, Gilbert RL, Beck JE. Intraoperative awareness in a regional medical system: A review of 3 years’ data. *Anaesthesiology*. 2007; 106: 269–274.

Pandit JJ, Cook TM. The NAP5 Steering Panel. NAP5. Accidental Awareness During General Anaesthesia. London. *The Royal College of Anaesthetists and Association of Anaesthetists of Great Britain and Ireland*. 2014.

Royal College of Anaesthetists. Patient information sheet. Section 8: Awareness during general anaesthesia. 2017. [www.rcoa.ac.uk/docs/Risk 8awareness.pdf](http://www.rcoa.ac.uk/docs/Risk%20awareness.pdf).

Sebel PS, Bowdle TA, Ghoneim MM, et al. The incidence of awareness during anesthesia: A multicenter United States study. *Anesthesia and Analgesia*. 2004; 99: 833–839.

2.5.9 Electroconvulsive Therapy (ECT) and Anti-psychotics – Amin Elkhawad

This topic could come up in the science structured oral exam where they may concentrate on the physiological changes that occur during ECT or pharmacology of psychoactive medications.

They may start with an introductory question.

What is ECT?

A simple answer is all that is required.

ECT is a treatment for severe depression and other severe psychiatric conditions. It involves passing an electrical current across the skull to induce a generalised tonic-clonic seizure. It is an effective treatment. However, its use is accompanied by public disquiet and the problem with autobiographical memory loss is a significant drawback.

Do you know any of the characteristics of the electric current used?

The current is an alternating current delivered as a pulsatile, square waveform. It has a current of 500 to 850 mA, which is applied through electrodes placed at specific locations on the head with right unilateral being the preferred placement. The energy used is 30–45 Joules and it is given over 0.5–1.5 seconds. Different devices have been used for administration of ECT which differ by the waveform of the stimulus delivered and whether they deliver a constant current, energy or voltage.

What are the indications for ECT in severe depression?

ECT is used in severe depression if other treatments have been unsuccessful, if a rapid response is required for life-threatening situations (e.g., not drinking/eating) as well as if the patient has a preference for this modality based on prior experiences.

Can you describe to me the physiological changes that occur during ECT?

These can be classified into systems to help structure your answer. Thinking of the effects of a generalised tonic-clonic seizure can also help you remember.

The induction of a seizure causes physiological responses in a number of systems.

Firstly, the central nervous system. The seizure starts with a short latent phase, which is followed by a tonic phase that lasts about 15 seconds. During this tonic phase there is general skeletal muscle contraction. This is followed by a clonic phase, which lasts 30–60 seconds. The EEG seizure activity continues beyond the motor seizure activity, which is 30% shorter. There may also be a post-ictal phase with confusion and agitation. Other CNS changes include an increase in cerebral blood flow and an increase in both intracranial pressure and intraocular pressure. The cerebral oxygen consumption also increases by up to a factor of four.

Secondly, there may be significant cardiovascular changes. Initially there may be a parasympathetic discharge following the passage of the current. This may be intense and can cause bradycardia, hypotension or even asystole. Atropine should always be readily available. This is followed by a sympathetic discharge, which starts as the clonic phase of the seizure begins. Adrenaline levels may rise 15 times higher than baseline and

noradrenaline levels three times higher, potentially causing tachycardia, hypertension and arrhythmias. The myocardial oxygen consumption also increases.

Good. What other systems are involved?

There are gastrointestinal changes associated with ECT. These include an increase in intragastric pressure, increased salivation and nausea and vomiting.

The musculoskeletal system is also affected by ECT. Uncontrolled muscular contractions may cause bony and musculoskeletal injury, for example vertebral fractures and joint dislocations. The jaw muscles are stimulated directly by the passage of current causing clenching of the jaw and possible injury to teeth, tongue, or other structures in the oral cavity. Therefore, pharmacological muscle relaxation and bite blocks are used in modern 'modified' approaches.

You have been allocated to administer anaesthesia for the electroconvulsive therapy (ECT) list. Describe your preoperative assessment.

Start by reassuring the examiner that you would take a thorough history and examination as for any anaesthetic but he or she wants you to concentrate on those points that are of particular relevance to ECT.

The preoperative assessment should include a detailed history and examination. There are some specific considerations in relation to ECT. The patient population can be elderly with numerous comorbidities.

A history of any cardiovascular disease should be sought. ECT should not be used in patients who have suffered a myocardial event within 3 months. The severity of any ischaemic heart disease should be evaluated by assessing exercise tolerance and frequency of symptoms. Severe ischaemic heart disease is a relative contraindication to ECT and therefore, if there are any concerns, should be investigated prior to starting on a course of ECT which may include a referral to cardiology as required.

In relation to the CNS, a history of any intracranial pathology is also important. ECT should not be administered to a patient who has had a cerebrovascular event within 3 months or who has raised intracranial pressure or a CNS mass lesion.

With regards to the musculoskeletal system, a history of osteoporosis or any other bone disease that increases the likelihood of fractures should be identified. ECT should probably be avoided in patients who have a high risk of fractures, but this needs to be discussed with the psychiatrist and the physician involved in the patient's care.

As with any patient a careful assessment of fasting status and risk of aspiration, for example a hiatus hernia, should be made.

Finally, glaucoma is a relative contraindication to ECT and the treatment should be discussed with an ophthalmologist if there are any concerns.

As well as informing the patients of the benefits and risks of ECT it is also important to inform them of the risks of not having ECT. If informed consent cannot be sought remember to seek out any advanced directives the patient may have.

During the preoperative assessment, it is important to remember that these patients may be unreliable historians, making the assessment difficult. It is often worth talking to nursing staff and other care givers for information and it is important to get hold of

medical notes to help identify and quantify any coexisting disease. It is worth remembering that these patients will often be having a course of ECT and will therefore receive a number of general anaesthetics in close succession, which may be a problem for frail patients.

Are there any specific drugs that the patients may be taking that may be relevant to us as anaesthetists?

Yes, and a detailed drug history should be taken. Firstly, all anticonvulsants should be withheld prior to ECT. Secondly, the patients are often taking psychoactive drugs that may interact with drugs given during anaesthesia. In particular, antidepressants such as monoamine oxidase inhibitors, tricyclic antidepressants and selective serotonin reuptake inhibitors.

Can you explain how monoamine oxidase inhibitors work?

There are two monoamine oxidase enzymes that these drugs inhibit; MAO-A and MAO-B. MAO-A is mainly intra-neuronal and degrades dopamine, noradrenaline and serotonin. Increased levels of these amine neurotransmitters are thought to elevate mood. MAO-B is extracellular and degrades other amines such as tyramine, tryptamine and phenylethylamine.

The older MAO inhibitors such as phenelzine are irreversible and block both MAO-A and -B. Moclobemide is reversible and selective for MAO-A and drugs such as selegiline, used in Parkinson's and severe depression, is a selective but irreversible inhibitor of MAO-B.

Why are they of concern to us as anaesthetists?

Monoamine oxidase inhibitors potentiate the actions of any indirectly acting sympathomimetics such as ephedrine and metaraminol. This can cause an exaggerated hypertensive response. Co-administration should therefore be avoided. Directly acting drugs such as noradrenaline and adrenaline should be used in small amounts if needed.

MAOIs also interact with piperidine derived opiates such as pethidine. This interaction may result in agitation, tachycardia, hyperpyrexia, muscle rigidity, hypertension and even coma. It is thought that this is due to excessive serotonin activity.

The newer drugs that are selective for MAO-A, cause less potentiation of amines. However, the drugs mentioned should still be avoided. Ideally, if elective surgery is planned, these drugs should be stopped two to three weeks beforehand. This should be done only after discussion with the patient's psychiatrist due to the risk of worsening depression.

Anaesthesia for ECT is often given in remote, isolated locations. What are the concerns with anaesthetising patients in such areas?

In these remote locations help and backup may not be readily available. There may not be critical care facilities close by and there may not be adequate equipment available to deal with an unforeseen event. The patients should therefore be assessed with special consideration made to their suitability for anaesthesia in a remote site. In general ASA grade 1 and 2 patients can be anaesthetised in remote sites. Patients graded ASA 3 or

more should be discussed with a consultant anaesthetist on a case-by-case basis. However, unexpected problems can also occur in ASA 1 and 2 patients. Resuscitation equipment should be available as well as a trained assistant and recovery facilities.

Describe how you would anaesthetise a patient for ECT.

It is a good idea to classify your answer into preoperative, intraoperative and postoperative management. If the examiner only wants you to discuss the perioperative management, he or she will guide you towards this.

Preoperatively I would take a detailed history and examination as I have already discussed. Any coexisting disease should be optimised. Sedative premedication should be avoided, in particular benzodiazepines, which increase the seizure threshold. Anticholinergics may be given to decrease the risk of bradycardia and salivation.

Resuscitation equipment should be available. Standard monitoring as outlined by the Association of Anaesthetists should be used. I would also ensure that I had a trained assistant.

If there were no concerns regarding the airway or risk of aspiration, I would preoxygenate the patient before inducing anaesthesia with a single minimal sleep dose of propofol. I would then maintain the airway with a facemask and a 100% O₂, hand ventilating as required. I would give a 0.5 mg/kg dose of suxamethonium to cause incomplete muscular paralysis and then insert a bite block, making sure that it is correctly placed, before allowing the psychiatrist to administer the stimulus. The doses of induction agent and suxamethonium should be recorded, as should the patient's response to them; this will allow the dose to be adjusted next time if needed. If I had any concerns regarding risk of aspiration, I would perform a rapid sequence induction using a 1 mg/kg dose of suxamethonium. Rocuronium and sugammadex may be an alternative if given at the appropriate dose.

I would observe the patient for signs of a fit and carefully monitor his or her cardiovascular response, treating any bradycardias with atropine or glycopyrrolate if required. I would continue to ventilate the patient until spontaneous ventilation returns.

Postoperatively, the patient should be recovered as normal until fully alert. Headaches are common postoperatively and should be treated with simple analgesics.

Good. We discussed the antidepressant drugs that patients undergoing ECT may be taking. What other mood altering drugs do you know of?

Although you may mention drugs such as the anti-psychotics, the examiner is likely to want you to discuss lithium, which has clinical implications in anaesthesia and intensive care.

Lithium is used to control mood in bipolar manic depression.

Tell me about lithium.

Lithium imitates the action of sodium and enters cells via fast, voltage gated channels. However, it cannot be pumped out by the sodium–potassium ATPase pump and accumulates in the cytoplasm. It is then thought to interfere with cyclic-AMP and inositol triphosphate second messenger systems. Lithium has a very narrow therapeutic index; therefore plasma levels should be measured regularly to ensure effective plasma levels of 0.5 to 1.0 mmol/L and avoid toxicity.

What are the adverse effects of lithium?

Lithium has a number of adverse effects including diarrhoea, vomiting, hypothyroidism and renal impairment. Inhibition of ADH may cause polydipsia and polyuria.

What signs and symptoms might make you suspicious of lithium toxicity?

Acute toxicity may present as ataxia, confusion, convulsions, arrhythmias or as a coma.

Lithium is also one of the most common causes for acquired nephrogenic diabetes insipidus leading to polydipsia and polyuria.

What are the important considerations when anaesthetising a patient on lithium?

The two main considerations are lithium toxicity and potential drug interactions. To prevent toxicity, plasma levels should be measured preoperatively as well as urea and electrolytes. Dehydration, the use of diuretics and hyponatraemia can potentiate toxicity. Therefore, it is important to maintain hydration and ensure electrolytes are normal prior to surgery.

The main drug interaction that is of concern to us as anaesthetists is that it prolongs the effect of all muscle relaxants. Care should also be taken when using NSAIDs as they may reduce lithium clearance and increase plasma levels.

Further Reading

Ferrier IN, Waite J. The ECT handbook. Cambridge: Cambridge University Press; 2019.

Guideline for the provision of anaesthesia services in the non-theatre environment. Royal College of Anaesthetists. March 2021.

National Institute for Health and Care Excellence. Depression in adults: treatment and management [Internet]. [London]: NICE; 2022 [updated 2016]. (Clinical guideline [NG22]). Available from: www.nice.org.uk/guidance/ng222

Reasoner J, Rondeau B. Anesthetic Considerations in Electroconvulsive Therapy. [Updated 2022 May 17]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-. Available from: www.ncbi.nlm.nih.gov/books/NBK576431/.

Uppal V, Dourish J, Macfarlane A. Anaesthesia for electroconvulsive therapy. *Continuing Education in Anaesthesia Critical Care and Pain*. 2010; 10(6): 192–196.

2.5.10 Epilepsy – Imran Mohammad and Stephen Pearson

A 28-year-old woman presents to the emergency department with generalised tonic–clonic seizures for the past 30 minutes. You have been called to help. What is your approach?

Epilepsy is important to the anaesthetist in clinical practice and therefore is likely to be covered in the Final FRCA. Epilepsy is a common comorbidity in our surgical patients, and we need to be aware of medication and drug interactions, the effects of our anaesthetic technique on postoperative seizures, and the intensive care management of status epilepticus.

This is status epilepticus and is a medical emergency. The definition of status epilepticus is a seizure lasting more than 5 minutes.

The immediate management consists of performing an ABCDE assessment and giving medication to terminate the seizure.

National Institute for Health and Care Excellence provides guidelines with a stepwise approach to managing status epilepticus.

Initial treatment consists of giving a benzodiazepine. In hospital, IV lorazepam is recommended, 0.1 mg/kg, up to a maximum of 4 mg. Local policies may allow alternatives such as diazepam, if lorazepam is unavailable. If IV access is unavailable, buccal, rectal or intramuscular benzodiazepine preparations are available but early intraosseous access should also be considered.

If 5–10 minutes after the first dose, the seizure has failed to terminate, a second dose of IV lorazepam should be given.

If the seizure does not respond to two doses of lorazepam, a second line agent should be used. Options include levetiracetam, phenytoin or sodium valproate. The choice of second line agent should be based upon patient factors and local policy.

An alternative second line agent can be used if the first medication is not effective.

Continued seizure activity despite second line agents should prompt induction of general anaesthesia. Historically this has been using sodium thiopentone but use of propofol is increasingly common.

Further management includes investigating the cause of the seizure. Venous blood should be taken for FBC, U&Es, glucose, antiepileptic drug levels (if appropriate). An arterial blood gas would be useful. Imaging, further investigations and treatment would be guided by the clinical presentation and patient's background. For example, if the patient was known to have a history of alcohol excess, IV thiamine and correction of glucose and electrolytes would be important. If the patient was pregnant, eclampsia would be managed with IV magnesium. EEG monitoring may be useful in conjunction with specialist input.

What factors may influence your choice for 2nd line agent?

Levetiracetam is safe in women of childbearing potential, does not require cardiac monitoring and has the fewest side effects and interactions of the antiepileptic agents. Routine drug level monitoring is not required and it has an oral bioavailability of close to 100% which reduces the chance of errors when changing formulation.

Phenytoin is safe in women of childbearing potential. It has been used historically; therefore clinicians are typically familiar with its use. IV infusions require cardiac monitoring. It carries a high risk of tissue necrosis in extravasation and as a cytochrome P450 enzyme inducer has many drug interactions.

Sodium valproate is contraindicated in women of childbearing potential and should be avoided in liver disease. As a potent inhibitor of the cytochrome P450 enzyme system it also has many drug interactions.

These three agents have been shown to have equivalent efficacy in terminating benzodiazepine refractory status epilepticus.

What are the complications of status epilepticus?

The complications can be divided into the systems of the body. Beginning with the central nervous system; cerebral hypoxia, oedema, haemorrhage and venous thrombosis

can occur. In the cardiovascular system, there may be hyper- or hypotension, myocardial infarction, arrhythmias, cardiogenic shock and cardiac arrest. In the respiratory system, complications include apnoea, respiratory failure, pulmonary oedema, aspiration and pneumonia. Metabolic complications include hyponatraemia, hypoglycaemia, hyperkalaemia, metabolic acidosis, acute tubular necrosis, acute hepatic necrosis and acute pancreatitis. Other complications include DIC, rhabdomyolysis, bone fractures and joint dislocations.

What is the differential diagnosis of epileptic seizures?

The differential diagnosis is broad and includes pseudo-seizures, arrhythmias, drug-induced seizures, hypoglycaemia, head injury, syncope, transient ischaemic attacks, narcolepsy and cataplexy.

Can you classify epileptic seizures?

There are many types of epilepsy. Most commonly they are classified by their clinical presentation as the management also follows this classification. They are divided into generalised and focal seizures. Generalised seizures are characterised by diffuse symmetric brain involvement and can be further broken down into inhibitory and excitatory. Inhibitory generalised seizures can be absence, petit mal or atonic. Excitatory seizures are the classic tonic, clonic and myoclonic variations.

In focal seizures, there is seizure activity restricted to discrete areas of the cerebral hemispheres. Consciousness is preserved in simple focal seizures whereas it is impaired in complex focal seizures.

Which drugs are used in preventing epileptic seizures?

For generalised seizures, sodium valproate is commonly used except for in women of childbearing age where lamotrigine or levetiracetam is recommended. Focal seizures are normally managed with lamotrigine, levetiracetam or carbamazepine. Ethosuximide is used for absence seizures. Other common antiepileptic medications include phenytoin, clobazam, topiramate and lacosamide.

Tell me about phenytoin

If asked about a drug, use a template for breaking down the information into easily memorable chunks. This also gives the impression of comprehensive knowledge about a drug. A convenient template is to use the headings presented in Sasada and Smith's Drugs in Anaesthesia and Intensive Care. These headings are uses, chemical presentation, main actions, mode of action, routes of administration/dose. Pharmacodynamics is divided into the relevant body systems such as CNS, CVS, metabolic etc., toxicity/side effects. Pharmacokinetics should be divided into absorption, distribution, metabolism, excretion, and any other special points.

Phenytoin is used for prevention and treatment of generalised tonic-clonic seizures. It is also used as an antiarrhythmic and neuropathic pain-modulating agent. The chemical class is a hydantoin derivative. In intravenous form it is presented as a clear, colourless solution of 50 mg/ml of phenytoin sodium. The main actions are anticonvulsant and anti-arrhythmic. Phenytoin works by inhibiting sodium and calcium influx during depolarisation, thereby exerting a membrane-stabilising effect.

The intravenous loading dose for the management of epilepsy is a slow bolus of 15–20 mg/kg. It is highly lipid-soluble and peak brain levels are achieved within 15 minutes. The maintenance dose is 100 mg 8-hourly.

Phenytoin has an anticonvulsant effect on the CNS by stabilising the neuronal membrane and preventing the spread of seizure activity. It exhibits class I anti-arrhythmic properties but may cause hypotension and arrhythmias itself. Metabolic effects include hyperglycaemia, hypocalcaemia, alterations in liver function tests and inhibition of ADH secretion. The side effects include gum hyperplasia, megaloblastic anaemia, nausea and vomiting, tremor and ataxia.

Phenytoin is 95% protein bound. It is metabolised in the liver, exhibiting zero order kinetics just above its therapeutic range. It is excreted as inactive metabolites by the kidney. It is a potent enzyme inducer increasing the metabolism of many drugs including carbamazepine, benzodiazepines and warfarin. Importantly phenytoin toxicity can be precipitated by metronidazole and isoniazid co-administration.

What non-pharmacological options are available for treating epilepsy?

In drug resistant epilepsy and specific epilepsy syndromes, a ketogenic diet may be recommended. Additionally, patients can be referred for resective epilepsy surgery or vagal nerve stimulators.

How would you manage a patient with epilepsy for elective surgery?

In addition to a standard preoperative assessment, specific information should be sought about associated comorbidities, seizure history and antiepileptic medications, including compliance. Requirements for specific investigations would be determined by the patient's comorbidities and type of surgery. A 12-lead ECG may be useful in patients taking carbamazepine or phenytoin due to risk of arrhythmias. Anticonvulsants may reveal anaemia, thrombocytopenia or leukopenia on full blood count. Electrolyte disturbances should be excluded. Routine testing of antiepileptic drug levels is not recommended unless there is a concern about compliance or toxicity. Airway assessment may identify gingival hyperplasia and poor dentition in patients taking phenytoin. Involvement with the patient's neurologist may be valuable in complex cases.

What are the effects of anaesthetic drugs on epilepsy?

Thiopentone and benzodiazepines are potent anticonvulsants. Although propofol is anticonvulsant, it is occasionally associated with abnormal movements which may mimic seizures. Etomidate is associated with more postoperative seizures and is less anticonvulsant than other induction agents which is why it is used in electroconvulsive therapy.

Ketamine may be proconvulsant at lower doses but it is anticonvulsant at anaesthetic doses.

Inhalation agents are anticonvulsant with the exception of enflurane which is pro-convulsant in higher concentrations. Laudanosine, a metabolite of atracurium, has epileptogenic potential but this is only demonstrated in animal studies. Other neuromuscular blockers are safe.

Further Reading

Carter, FRCA Eleanor L, Adapa, MD PhD
FRCA Ram M. Adult epilepsy and anaesthesia. *BJA Education*. 2015; 15, (3): 111–117.

National Institute of Health and Care Excellence. *Epilepsies in children, young people and adults* [NICE Guideline NG217]. 2022. www.nice.org.uk/guidance/ng217.

2.5.11 Spinal Surgery and Neuromuscular Monitoring – Alexandra K Freeman

You are asked to anaesthetise a 55-year-old patient for a thoracic spine fusion. The surgeon informs you that a neurophysiologist will be present to undertake intraoperative neuromonitoring.

What is intraoperative neuromonitoring?

Intraoperative neuromonitoring includes a number of modalities used to monitor the integrity of neural pathways during surgery. It is used to detect impending neurological injury, allowing prompt intervention in order to prevent permanent deficit.

Which procedures may require intraoperative neuromonitoring?

Intraoperative neuromonitoring may be used during any surgical procedure in which there is a risk of damage to one or more important territories within the brain, spinal cord or peripheral nerves. This would include:

- Intracranial surgery, such as resection of intracranial tumours or vascular lesions;
- Spinal surgery, such as resection of spinal tumours, deformity correction or spinal cord decompression;
- Aortic surgery where there is risk of spinal cord ischaemia; or
- Any surgery in close proximity to cranial or peripheral nerves, such as parotid or thyroid surgery.

Which modalities of intraoperative neuromonitoring are you aware of?

The different modalities can be divided into those which measure spontaneous potentials and those which measure evoked potentials. Techniques which measure spontaneous potentials include electroencephalography (EEG) and electromyography (EMG). Those which measure evoked potentials include somatosensory evoked potentials (SSEPs), motor evoked potentials (MEPs), brainstem auditory evoked potentials (BAEPs) and visual evoked potentials (VEPs).

Another method of intraoperative neuromonitoring, which does not require electro physiological equipment, is the Stagnara Wake Up test. This is a test for voluntary motor function. It involves waking the patient from anaesthesia during the procedure and asking them to move their feet. Although relatively simple, it does not detect the onset of neurological injury and can miss subtle changes; for these reasons its use has been largely superseded by electrophysiological methods.

Tell me a bit more about somatosensory evoked potentials and how they are monitored?

This question may seem difficult as they are not something anaesthetists monitor in our day-to-day practice; however, the concepts are relatively simple. It would be helpful to have seen at least one case requiring monitoring of somatosensory and motor evoked potentials prior to the exam.

Somatosensory evoked potentials (SSEPs) monitor somatosensory pathways of the dorsal columns and spinothalamic tracts. Monitoring involves transcutaneous stimulation of peripheral nerves (commonly posterior tibial, ulnar or median nerves) and monitoring of the sensory cortex via scalp electrodes. SSEPs are small amplitude (1–2 μV , compared to 100 μV for an EEG). Multiple stimulations are, therefore, made and an average is found. A reduction in amplitude of 50% or an increase in latency of 10% are commonly used as markers of neurological compromise.

What about motor evoked potentials?

Motor evoked potentials (MEPs) monitor motor pathways of the corticospinal tracts. Monitoring involves stimulation of the motor cortex via scalp electrodes, or by direct stimulation, and monitoring of compound muscle action potentials via a needle placed within the muscle. Commonly used muscles include adductor hallucis, tibialis anterior or vastus medialis.

When compared to SSEPs, MEPs are more sensitive and more rapid at demonstrating alterations in response. A reduction in amplitude of 50% or more may be considered significant.

How do common anaesthetic drugs affect SSEPs and MEPs?

Many drugs used in routine anaesthetic practice affect monitoring of SSEPs and MEPs and it is pertinent to inform the neurophysiologist when any of these are given.

Volatile anaesthetic agents

All of the volatile anaesthetics cause a dose-dependent reduction in MEP and SSEP amplitudes and an increase in SSEP latencies. Although usually avoided, desflurane and sevoflurane may be used at a concentration up to 1.0 MAC. Nitrous oxide significantly decreases SSEP amplitudes and should be avoided.

Intravenous anaesthetic agents

Propofol causes a dose-dependent reduction in MEP and SSEP amplitudes but to a lesser degree than volatile agents. For this reason, total intravenous anaesthesia (TIVA), in conjunction with cerebral function monitoring, is usually the anaesthetic technique of choice. Thiopentone has minimal effect on SSEPs or MEPs at anaesthetic doses. Ketamine and etomidate both increase the amplitude of SSEPs and MEPs.

Muscle relaxants

Muscle relaxants improve the measurement of SSEPs, which are of very small amplitude and therefore susceptible to movement artefact, but impair the measurement of MEPs, which rely on integrity of the motor pathways. They must, therefore, be used cautiously.

Opioids

Opioids have minimal effect on SSEPs and MEPs. For this reason, remifentanyl is an excellent adjunct to a TIVA regimen, thereby reducing the dose requirement of propofol and alleviating the need for repeated doses of muscle relaxant.

Benzodiazepines

Benzodiazepines have minimal depressant effects on SSEPs and MEPs.

Are you aware of any physiological variables which may affect SSEPs and MEPs?

Neural tissue has a high oxygen requirement and, therefore, any physiological change which impairs oxygen delivery can affect monitoring of evoked potentials. This includes mean arterial pressure, partial pressure of oxygen and carbon dioxide, haemoglobin concentration and temperature.

Mean arterial pressure is an important determinant of spinal cord perfusion and MAP values <60 mmHg increase the risk of spinal cord injury. Hypotension reduces amplitudes of both SSEPs and MEPs, with a greater effect on MEPs due to the perfusion of the corticospinal tracts by a single anterior spinal artery.

Hypoxaemia causes a decrease in amplitude and increase in latency of SSEPs. Hypocapnoea also decreases SSEP amplitudes due to its effect on cerebral vasoconstriction.

Anaemia (specifically haematocrit $<15\%$) causes a decrease in amplitude and increase in latency of SSEPs.

Hypothermia causes a decrease in amplitude of SSEPs and MEPs and an increase in latency of SSEPs. Mild hyperthermia (up to 39°C) decreases SSEP latencies; however, above this temperature latencies are prolonged and amplitudes are reduced.

What should be done if a significant change in the evoked potentials is detected intraoperatively?

The signal changes would be detected by the neurophysiologist who would alert the team. A collaborative response from the surgeon, anaesthetist and neurophysiologist would be required, following a Standard Operating Procedure, if available.

The surgeon should stop manipulation, relieve any compression and consider revising any recent instrumentation (e.g. removal of screws), if safe to do so. The anaesthetist should assess for and treat any pharmacological or non-pharmacological cause, by ensuring adequate MAP, PaO_2 , PaCO_2 , haemoglobin and temperature, in addition to ensuring an appropriate depth of anaesthesia and degree of neuromuscular block. The neurophysiologist should check all electrodes and repeat the measurements to exclude erroneous readings and to confirm improvement following any surgical or anaesthetic interventions.

What if there was no improvement with the interventions you have mentioned?

If the neurophysiologist had confirmed that the readings were accurate but there were still concerns about the measurements, experienced colleagues should be consulted. All of the anaesthetic and surgical factors mentioned should be revisited. A dose of steroid

(e.g. 30 mg/kg IV methylprednisolone) could be administered. At that point, the surgical team would need to consider whether it was appropriate to continue with the procedure as planned or to limit the extent of surgery.

You mentioned electromyography. What is this and how is it measured intraoperatively?

Electromyography (EMG) involves continuous monitoring of a specific muscle innervated by a single nerve or nerve root. The muscle activity is used as a surrogate marker for the integrity of the innervating nerve. It requires placement of multiple needle electrodes into the muscle, which monitor spontaneous and induced activity. EMG may be used during spinal surgery below the level of the spinal cord (e.g. lumbar fusion) or surgery in close proximity to cranial or peripheral nerves (e.g. parotid or thyroid surgery).

Finally, tell me about intraoperative EEG monitoring.

Intraoperatively, electroencephalography (EEG) can be used to monitor cerebral perfusion and depth of anaesthesia. It requires placement of a number of scalp electrodes which measure cerebral cortical activity and present this as a series of waves (called an encephalogram). The EEG waves may be classified according to their frequency. Beta waves (12–25 Hz) and alpha waves (8–12 Hz) are higher frequency and predominate during periods of wakefulness. Theta waves (4–8 Hz) and delta waves (1–4 Hz) are lower frequency and predominate during periods of unconsciousness. Furthermore, different anaesthetic agents produce specific patterns of EEG waveforms. With very deep levels of anaesthesia, the EEG may exhibit burst suppression.

The interpretation of raw EEG waveforms requires specific skills and, therefore, processed EEG monitors (such as BIS and entropy) are frequently employed. These monitors produce indices in the range 0–100 to indicate levels of consciousness. A range of 40–60 is usually considered appropriate for surgical anaesthesia. Values <40 indicate increasing frequency of burst suppression.

2.5.12 Anaesthesia for Awake Craniotomy – Alexandra K Freeman

You are asked to anaesthetise a 47-year-old patient for an awake craniotomy and resection of a primary brain tumour.

What are the indications for an awake craniotomy?

Awake craniotomy is an important technique for facilitating removal of tumours and vascular lesions near critical areas of the brain (including the speech and language centres, motor and sensory cortex). Monitoring of special skills during the procedure reduces the risk of postoperative neurological deficit while permitting more extensive resection. It may also be used in epilepsy surgery and deep brain stimulation.

Can you list any contraindications to an awake craniotomy?

The contraindications may be divided into patient factors and surgical factors.

Patient factors contraindicating an awake procedure include: patient refusal, severe anxiety, uncontrolled seizures, an inability to lie flat or still (e.g. due to neurological,

respiratory or musculoskeletal comorbidities) or an inability to follow commands (e.g. acute confusion, learning difficulties or a language barrier).

Surgical contraindications include procedures requiring the prone position (e.g. resection of low occipital tumours).

What are your specific considerations during the preoperative assessment of this patient?

Remember to discuss general considerations relating to any neurosurgical patient, as well as those specific to an awake craniotomy.

My preoperative assessment would include a thorough history, examination and review of investigations. In addition to general neurosurgical considerations, I would want to assess the patient's suitability for an awake procedure.

Specific aspects of the history would include the indication for surgery, presence of neurological deficits, features of raised intracranial pressure and, if present, seizure type and frequency. I would assess for any comorbidities which may affect my anaesthetic, specifically cardiorespiratory comorbidities, and those which may contraindicate an 'awake' procedure, including an inability to lie flat and still or an inability to follow commands. My drug history would assess for the use of steroids (including dose and duration) which may require perioperative supplementation, antiepileptics (which should be continued perioperatively), and anticoagulant agents (which may affect surgical haemostasis).

My examination would include a gross neurological assessment and documentation of any deficit in order to monitor changes in the postoperative period, as well as a meticulous airway assessment, which is particularly important for an awake craniotomy as the patient may be re-anaesthetised while on the operating table.

The investigations I require would be guided by my history and examination but would include blood tests (including a full blood count, coagulation studies, liver function tests, urea and electrolytes) and brain imaging (CT or MRI).

After my assessment, I would ensure that the patient had been appropriately counselled about the procedure. This would most likely have happened over several visits by both the surgical and anaesthetic teams and may have included a visit to the operating theatre.

What techniques are you aware of for providing anaesthesia for an awake craniotomy?

While the examiner will not expect you to be competent to undertake this anaesthetic independently, it will be pertinent to demonstrate that you are aware of the options. Try to see at least one awake craniotomy prior to the exam.

I am aware of several different anaesthetic techniques for awake craniotomy, all of which aim to provide a comfortable, still and compliant patient. The procedure can be divided into three stages: craniotomy, brain mapping and resection, and closure. The most stimulating parts of the procedure are the craniotomy and closure, which usually require either sedation or general anaesthesia. During brain mapping and resection, the patient will need to be 'awake' in order to follow instructions.

The possible anaesthetic techniques include asleep-awake-asleep (where the patient is anaesthetised for the craniotomy, awoken for the brain mapping and resection, and then

re-anaesthetised for closure), asleep-awake (where the patient is awoken for the resection but not re-anaesthetised for closure) or awake throughout (where the patient receives only local anaesthesia with/without sedation). In all cases a scalp block is usually performed to provide analgesia for the craniotomy site.

What are the general principles when providing anaesthesia for an awake craniotomy? How are these achieved?

General principles for any craniotomy include:

- Maintaining cerebral perfusion pressure
- Avoiding raised intracranial pressure
- Haemodynamic stability
- Seizure prophylaxis.

Cerebral perfusion pressure is the difference between mean arterial pressure and intracranial pressure. Intracranial pressure is maintained through controlled ventilation (avoiding hypercapnoea and hypoxia), avoiding sympathetic stimulation (through adequate depth of anaesthesia and analgesia) and preventing coughing and straining. Haemodynamic stability is maintained through invasive blood pressure monitoring and the use of vasopressors to achieve a target mean arterial pressure. Seizure prophylaxis is achieved through the use of antiepileptic agents (e.g. 1 g levetiracetam IV at the time of induction).

Specific anaesthetic principles for an awake craniotomy include:

- The ability to rapidly control conscious level
- Patient comfort and cooperation
- Avoidance of hypoventilation and airway obstruction.

Rapid control of conscious level and smooth emergence from anaesthesia can be achieved through the use of short-acting, easily titratable drugs. Total intravenous anaesthesia with propofol and remifentanyl is the technique with which I am most familiar. Other agents include infusions of alpha-2-receptor agonists (clonidine or dexmedetomidine). Patient comfort is achieved through adequate analgesia (usually in the form of a scalp block and local anaesthetic infiltration by the surgeons), judicious use of antiemetics, careful positioning on the operating table, ensuring the face is free from drapes and a calm environment during awakening. Cooperation can be achieved through careful patient selection and preoperative counselling. Hypoventilation and impending airway obstruction can be detected by visualising the patient and using a capnograph mask.

You mentioned providing a scalp block to facilitate analgesia. Please could you tell me how you would perform this?

Don't forget to start your answer by explaining that you would obtain informed consent, ensure that there were no contraindications to the procedure, prepare all your equipment and drugs (including emergency drugs), site an IV cannula, attach monitoring and ensure appropriate sterility. As with all regional techniques, never forget to mention 'stop-before-you-block'.

I would perform the scalp block after induction of general anaesthesia (or after commencing sedation) but prior to insertion of the Mayfield pins. After appropriate

preparation of the patient and my equipment, I would don sterile gloves and clean the skin with chlorhexidine. Immediately prior to performing the block I would carry out the 'stop-before-you-block' checks and re-confirm the patient details, procedure and block site against the consent form.

Using a 25-gauge needle I would infiltrate local anaesthetic to seven nerves on each side of the scalp, four of which are branches of the trigeminal nerve and three of which are branches of the C2 and C3 spinal nerves. The nerves include:

- Supraorbital nerve which is blocked by a subcutaneous injection over the supraorbital notch;
- Supratrochlear nerve which is blocked at the same time as the supraorbital nerve by continuing the subcutaneous injection 1 cm medially;
- Zygomaticotemporal nerve which is blocked by injecting from the lateral supraorbital margin to the zygomatic arch, deep and superficial to the temporals muscle;
- Auriculotemporal nerve which is blocked by a subcutaneous injection immediately above the temporomandibular joint, 1–2 cm anterior to the tragus (palpating the superficial temporal artery to avoid intravascular injection);
- Greater auricular nerve which is blocked by a subcutaneous injection 1–2 cm behind the ear, at the level of the tragus;
- Greater and lesser occipital nerves which are blocked by infiltrating along the superior nuchal line between the mastoid and the external occipital protuberance.

**The patient develops a generalised seizure during cortical mapping.
What would be your management?**

Intraoperative seizures are an anaesthetic emergency. I would alert the theatre team and call for help. My first intervention would be to request for the surgeon to irrigate the brain with ice-cold saline; this would usually terminate the seizure. If this was unsuccessful, I would administer a benzodiazepine (e.g. 4 mg IV lorazepam) or antiepileptic agent (e.g. 1 g IV levetiracetam). If the seizure continued despite drug treatment, I would re-anaesthetise the patient.

2.6.1 Physiological and Anatomical Changes of Pregnancy – Jade A Loughran and Sarah F Bell

What can you tell me about the physiological and anatomical changes of pregnancy?

A structured approach is vital to ensure that you do not forget any of the key points. It may be worth making an opening statement explaining the reason for all of these changes!

The physiological changes of pregnancy are aimed at supplying oxygen and nutrients to the increased demand of the uteroplacental unit and fetus. The adaptations are also key in preparing the mother for delivery. The changes may be divided into the body systems:

Firstly the cardiovascular system. These occur very early in pregnancy, leading to a hyperdynamic circulation. From the first trimester there is an expansion of plasma volume of about 50%. Red cell volume also increases, but due to the relatively greater increase in plasma volume a dilutional anaemia develops. Hormone changes cause peripheral vasodilation which leads to a fall in systemic vascular resistance. In order to meet the maternal and fetal demands the cardiac output increases by 40%. This is due to both an increase in heart rate and stroke volume. The increase in stroke volume occurs via increased preload due to increased blood volume. Heart rate increases, but a recent multicentre observational study showed that this increase is modest. Both systolic and diastolic blood pressure fall only very slightly between 12 and 19 weeks gestation, then rise above the 12 week values by term. This fall in blood pressure activates the renin-angiotensin-aldosterone system, leading to water retention and an increase in plasma volume. Volume expansion causes cardiac dilatation and hypertrophy, leading to ECG changes of left axis deviation and ST segment changes.

Another important cardiovascular effect is that of aortocaval compression. The gravid uterus compresses the vena cava, reducing venous return. It can reduce aortic blood flow, particularly when the parturient is in the supine position. This effect can have severe implications for the uteroplacental unit which does not have a system of autoregulation and so relies on pressure to maintain blood flow. In order to decrease the effects of aortocaval compression we use a tilt or wedge when the pregnant woman is supine.

How much weight would you expect a woman to gain during a normal pregnancy?

The examiner may interrupt if he feels you are performing well and he wants to probe your depth of knowledge further.

The weight gain varies tremendously but average values are approximately 12 kg. This is due to the combination of increase in tissue mass of the breasts, placenta, uterus and fetus and the increase in plasma and interstitial fluid.

Can you now describe the changes to the respiratory system?

There are many changes so try and remember as much as you can of this important topic. If the examiner allows you to continue you can indicate the implications to the anaesthetist.

The oxygen consumption of a pregnant woman increases by 20% to 40% at term. The minute ventilation increases by 45% to meet this increased demand. This is achieved predominately by an increase in tidal volume rather than respiratory rate and is thought to be caused by the stimulatory effects of progesterone on the central respiratory neurons. The upper limit (97% confidence interval) for respiratory rate of 22 breaths/min is important when considering an unwell patient during pregnancy.

The increased minute ventilation causes a mild respiratory alkalosis, which shifts the oxy-haemoglobin dissociation curve to the left, but the coinciding increase in maternal 2,3DPG offsets this potential deleterious effect. The upward displacement of the diaphragm by the uterus causes a decrease in the functional residual capacity of up to 25%. The combination of reduced FRC and increased oxygen consumption leads to an increased risk of hypoxaemia. The decreased FRC also leads to a more rapid onset of the volatile agents. Tracheal intubation of a pregnant woman can be more challenging due to upper airway capillary engorgement and oedema, breast enlargement and possible laryngeal distortion from cricoid pressure when the woman is in the tilted position.

Can you tell me how pregnancy alters the gastrointestinal system?

The gravid uterus elevates the stomach which increases intragastric pressure. Progesterone acts to decrease the lower oesophageal sphincter tone. This leads to fall in the barrier pressure (the difference between the lower oesophageal sphincter tone and the gastric pressure). Gastric motility is normal during pregnancy but reduced during labour, especially when opioid analgesia is administered.

How do these changes affect women?

Up to 70% of women suffer from gastro-oesophageal reflux during pregnancy due to reduced barrier pressure.

What are the anaesthetic implications?

There is an increased risk of aspiration if general anaesthesia is needed. Women should receive antacid prophylaxis during labour if there is any increased risk of emergency caesarean section. A prokinetic may also be given. Prior to induction of general anaesthesia, 30 ml of 0.3 M sodium citrate is given to neutralise acidic gastric contents. Furthermore a rapid sequence induction technique is employed in an attempt to avoid airway soiling by gastric contents.

How does pregnancy alter renal function?

The enlargement of the uterus leads to dilation of the ureters and calyx with reduced bladder capacity. Due to increased renal plasma flow, the glomerular filtration rate increases, and creatinine and urea levels fall. Glycosuria is commonly observed.

What haematological changes occur?

The woman becomes relatively hypercoagulable during pregnancy to reduce blood loss during labour. This is achieved by an increase in the concentration of fibrinogen and all of the coagulation factors except factor XIII. There is also a reduction in the activity of the natural anticoagulants. Enhanced platelet turnover occurs, and thrombocytopenia is seen in approximately 1% of pregnant women.

How does this affect your management of a woman after a lower segment caesarean section (LSCS)?

Any woman who receives a LSCS should have postoperative prophylactic anticoagulation to reduce the possibility of DVT or PE.

How does the WCC change during pregnancy and why is this important?

The leukocyte count increases slightly during pregnancy and then markedly during labour with levels reaching about $15\text{--}20 \times 10^9/\text{L}$. This is important to consider when attempting to use the white cell count as a marker for sepsis (e.g. when deciding whether to perform an epidural) as it may be unreliable. Full assessment for evidence of infection or sepsis is therefore necessary.

Do the plasma proteins change during pregnancy?

Yes, the concentration of albumin falls, but globulin and fibrinogen increase. The overall total protein level falls leading to a reduction in oncotic pressure. Drug binding is altered and free drug concentrations may increase if normally bound to albumin. Plasma concentration of pseudocholinesterase is reduced by approximately 20% and may therefore prolong the action of suxamethonium.

Do you know of any endocrine changes of pregnancy?

The thyroid gland increases in size but the mother usually remains euthyroid. Plasma corticosteroid levels increase 3–5 times and ACTH secretion is increased. The pituitary gland becomes enlarged, predominantly due to the increased activity of the prolactin secreting lactotropic cells, thus making it more susceptible to falls in perfusion due to its portal blood supply.

Women can become resistant to the effects of insulin and gestational diabetes can develop.

How is the musculoskeletal system affected?

The effects of progesterone and relaxin act to increase ligamentous laxity and increase the lumbar lordosis. It is therefore important to take care when positioning the patient.

Can you describe to me any of the central nervous system effects of pregnancy?

The pregnancy hormone progesterone and the beta endorphins released during labour may act to reduce MAC by as much as 30%. They also increase sensitivity to both sedatives and hypnotics. Venocaval compression caused by the uterus results in distension of the epidural venous plexus. This can lead to an increased risk of intravascular injection during regional anaesthesia and contributes to the enhanced spread of local anaesthetic due to the reduced capacity of the epidural space itself. The sympathetic nervous system activity is increased during pregnancy in order to produce lower limb vasoconstriction and counteract the effects of aortocaval compression.

Can you describe the innervations of the bladder, uterus, vagina and cervix?

The bladder has sympathetic innervation derived from roots T11 to L2 and parasympathetic innervation from S2 to S4. The uterus receives sympathetic innervation from the inferior hypogastric nerves, and parasympathetic innervation from S2 to S4, while the broad ligament receives its nerve supply from roots T10 to L1. The vagina and cervix are innervated via the pudendal, genitofemoral, ilioinguinal and sacral nerves from roots S2–4.

What level sensory block do you therefore need for a caesarean section using central neuraxial blockade?

The sensory block should be at or above T5 for light touch.²

What are the effects of maternal pain on the fetus and the mother?

Pain will cause sympathetic nervous system stimulation. This can lead to diversion of blood flow away from the uteroplacental unit. Since there is no autoregulation in this organ, the pain will directly affect the fetal blood supply. Pain can also cause a metabolic acidosis and an increase in plasma cortisol and endorphins.

Further Reading

Green LJ, Mackillop LH, Salvi D, Pullon R, Loerup L, Tarassenko L et al. Gestation-specific vital sign reference ranges in pregnancy. *Obstetrics and Gynecology*. 2020; 135(3): 653–664.

Plaat F, Stanford SER, Lucas DN, Andrade J, Careless J, Russell R et al. Prevention and management of intra-operative pain during Caesarean section under neuraxial anaesthesia: A technical and interpersonal approach. *Anaesthesia*. 2022; 77(5): 588–597.

2.6.2 Anaesthesia in Early Pregnancy – Farzad Saadat and Sarah F Bell

You may be asked about anaesthesia in early pregnancy within a number of different clinical scenarios. The examiner will want to know that you recognise the key differences that occur in pregnant patients compared to the non-pregnant population.

You are asked to anaesthetise a 35-year-old, 24-week primigravida woman who has acute appendicitis. She is otherwise fit and well and has not had any complications

during the pregnancy so far. She was admitted yesterday with abdominal pain, fever and a leucocytosis on her full blood count. The surgeons are keen to take her to theatre for an emergency appendectomy.

Can you briefly summarise the main issues to the anaesthetist regarding this case?

The main issues are that the woman has an acute abdomen requiring urgent surgery and that she is in the second trimester of pregnancy.

So what information would you try and obtain at the pre-op visit?

In my pre-op assessment, my history would be structured towards the following areas: confirmation of diagnosis, full anaesthetic history, acute state given the current illness and finally, obstetric history. I would consult the patient's notes and perform an examination to further establish the degree of any current sepsis.

In taking the history, I would pay particular attention to the presenting complaint. I would want to know how and when the pain had started, where it was located, whether it radiated, its severity and character, whether there were any exacerbating, relieving or associated factors. I would enquire about the patient's ability to eat and drink recently and whether she had had any vomiting or diarrhoea. I would also ask about fevers and whether the patient had experienced any of these symptoms previously. I would enquire about the patient's past anaesthetic and medical history and take a full systems history. I would then ask about medications, allergies and dentition. Regarding the pregnancy I confirm her gestational age and due date, and would enquire about any complications so far such as gestational diabetes, hypertension or cholecystitis of pregnancy.

I would perform bedside examination of the cardiovascular and respiratory system, as the physiological changes of pregnancy can unmask previously undiagnosed disease.

The patient is worried about the effects of drugs and surgery on the development of her baby? How would you counsel this woman on the risks of surgery in pregnancy?

The areas of concern of surgery during pregnancy are those of teratogenesis, risk of miscarriage, or risk of early labour. As the pregnancy is beyond 8 weeks, there is little risk of teratogenesis. In addition, there is no strong evidence of risk following exposure to anaesthetic agents, even in the first trimester.

There is a small increase in risk of miscarriage following surgery. However, as she is in her second trimester, this is the most favourable period to have non-obstetric surgery, as the risk of miscarriage is low, while still having good access to the abdominal organs. In addition, as she is becoming unwell from her appendicitis, the risk, to her and her baby, of not having surgery far outweigh the surgical risk.

What would you look for in your examination of this woman?

On examination I would look to assess the degree of dehydration, confirm the diagnosis and decide whether the patient was septic and/or in shock. I would therefore look at the observation chart to review the respiratory rate, heart rate, blood pressure, temperature, urine output and pain score. I would then examine the cardiorespiratory system and the

abdomen. While talking to the woman I would assess her conscious level and pain score. I would check whether the obstetricians had performed a vaginal examination and look in the notes to see their examination findings.

What investigations would you want to review before taking this woman to theatre?

I would want to have confirmation of the pregnancy, either by urine or blood test or by ultrasound scan. I would perform urinalysis and review the full blood count, urea and electrolytes, liver enzymes, amylase and glucose level. I would also ensure that a blood sample had been sent for group and save. Depending on the clinical picture an ECG, arterial blood gas and blood cultures might be required.

What action would you take prior to taking this woman to theatre?

Prior to taking this woman to theatre I would want to pre-optimize her condition as much as possible. This would require large bore intravenous access followed by fluid resuscitation, antibiotics and analgesia. Depending on the severity of her condition this might need to be done in recovery or the high dependency unit. Invasive arterial and central venous pressure monitoring might be required if the patient were in septic shock. The duration of time taken to pre-optimize that patient needs to be balanced against the need to take the patient to theatre to remove the source of sepsis.

I would also want to inform both the obstetricians and the neonatologists of the patient and her condition because there is a risk that she might develop premature labour either due to the sepsis or the operation. Fetal heart rate monitoring before and after the surgery should be discussed, and is often reassuring to the patient. In addition, I would inform the consultant anaesthetist on-call of the patient's condition.

Premedication would be indicated in this case. I would administer omeprazole (orally or IV, depending on patient tolerance) and sodium citrate 30 ml of 0.3 M solution in order to reduce the volume and acidity of the gastric contents. I would ensure that an experienced surgeon was available to perform the procedure since this may be technically challenging. I would also ask my assistant to have available the difficult airway equipment.

Why might intubation be more difficult in this patient?

Pregnant women have increased breast size, chest wall diameter, nasal engorgement and possibly laryngeal oedema which can all make intubation more challenging. They need to be positioned in a left lateral tilt to avoid aortocaval compression. This should be considered by the anaesthetic assistant performing cricoid pressure so that the view at laryngoscopy is not impaired. Lower oesophageal sphincter tone is reduced, causing an increased risk of reflux. Furthermore from 6 weeks gestation the increase in oxygen consumption and fall in oxygen reserve leads to a faster onset of hypoxia.

Why is the oxygen reserve lower?

The upwards displacement of the diaphragm causes a reduction in the functional residual capacity. Closing capacity can then encroach on FRC particularly in the supine position. The oxygen reserve is therefore reduced, even after careful de-nitrogenation.

What are the effects of progesterone on the respiratory system? Would they affect your general anaesthetic?

The hormone progesterone mediates tracheal and bronchial smooth muscle relaxation and so causes an increase in dead-space. Progesterone also sensitises the respiratory centre to changes in the partial pressure of carbon dioxide. This, coupled with the increase in carbon dioxide production occurring during pregnancy leads to an increase in the minute volume (mainly by increases in tidal volume). The pregnant woman develops a respiratory alkalosis due to the increase in minute volume. During an anaesthetic this normal physiological adaptation should be maintained.

What might be the cardiovascular effects of pregnancy on this woman? How might they affect your general anaesthetic?

In addition to the effects of aortocaval compression, the 24 week parturient will have a significantly raised plasma volume and cardiac output can increase by 50%. Any fall in blood pressure should be treated promptly as this will also reduce placental blood pressure.

How might the pregnancy alter the effects of the induction agents, volatile agents and neuromuscular blockers?

Pregnancy is associated with lower anaesthetic requirements. The exact mechanism is unknown. The minimum alveolar concentration for the volatile agents is reduced by 30% by 12 weeks gestation. The dose of intravenous induction agents is also reduced. In contrast, the effects of suxamethonium can be prolonged by the reduced concentration of plasma cholinesterase, but in reality this is offset by the increase in volume of distribution so minimal change in duration of action is usually observed. Of note, I would avoid the use of sugammadex for reversal of neuromuscular blockade, due to concerns around progesterone binding and a lack of data in pregnant populations.

What might be the effects on the renal system?

The increase in glomerular filtration rate leads to lower plasma concentrations of urea and creatinine. This should be considered when reviewing the patient's renal function with regards to dehydration, sepsis and drug dosing.

How would you anaesthetise this woman?

I would prepare all my emergency equipment, drugs and check my machine prior to the patient entering. I would anaesthetise her in the operating theatre. I would ensure that I had suction at hand and a trained assistant who is able to perform cricoid pressure. I would then obtain mandatory monitoring as per the AAGBI guidelines. I would ensure that the woman was placed in a left lateral tilt position. I would preoxygenate the woman with 100% oxygen for 3 minutes with a tight-fitting facemask. I would then ask my

assistant to put on cricoid pressure and induce anaesthesia with propofol and 2 mg/kg of suxamethonium. After 30 seconds I would intubate the patient with an appropriately sized cuffed oral tracheal tube. I would confirm correct positioning of the tube visually, by auscultation and by capnography. Then I would maintain anaesthesia using sevoflurane, oxygen and air, bearing in mind the reduction in minimum alveolar concentration associated with pregnancy. I would consider use of an EEG monitor to avoid over-anaesthetising and I would use a nerve stimulator to monitor neuromuscular function, since plasma cholinesterase enzyme activity may be impaired and cause prolonged effects of suxamethonium. With regard to analgesia, I would give IV paracetamol and increments of morphine. I would also ask the surgeons to infiltrate the wound with levobupivacaine. Non steroidal anti-inflammatory drugs should be carefully considered due to the risk of premature closure of the ductus arteriosus and so I would avoid their use in this case.

What would be your postoperative plan for this patient and where would you manage her?

I would ensure that the patient was comfortable, conscious and that her observations were stable prior to discharge from the recovery room. Depending on the extent of the surgery a morphine PCA might be required for postoperative analgesia in addition to regular paracetamol. I would contact the obstetric team to review the patient and ensure the well-being of the fetus. I would also discuss thromboprophylaxis with the surgical team.

The patient should be managed on the high dependency unit if there are concerns regarding her fluid balance or cardiorespiratory stability. If she were stable she could be nursed on an obstetric ward provided the nursing staff had some surgical experience or vice versa on a surgical ward. She would need to be regularly reviewed by both the obstetric and surgical teams.

Finally, can you tell me when anaesthetic drugs are most likely to have teratogenic effects?

The 15th–56th days of gestation are when the fetus is most vulnerable. Whenever possible, surgery should be delayed until the second trimester. Elective surgery should not be performed at all during pregnancy.

Further Reading

Haggerty E, Daly J. Anaesthesia and non-obstetric surgery in pregnancy. *BJA Education*. 2021; 21(2): 42–43.

2.6.3 Complications of Pregnancy – Farzad Saadat and Sarah F Bell

In complications of anaesthesia in pregnancy, be prepared for questions about common complications, or less common, but severe problems, such as maternal cardiac arrest.

You are called by a midwife to the labour ward to review a 31-year-old woman complaining of sensory loss in her left leg. She is recovering having delivered a healthy baby one day previously. The midwife is concerned as this woman had an epidural throughout labour and her delivery and thinks the epidural may be the cause of her loss of sensation. What are the possible causes of sensory loss in this patient?

Postnatal neurological problems can be due to obstetric causes, such as pregnancy or labour, anaesthetic causes, such as a complication of neuraxial anaesthesia or other pre-existing medical conditions. The majority of nerve damage cases are secondary to the mechanics of labour or fetal pressure on the nerves. However, I would take a careful history and examination before ruling out an anaesthetic cause, given the potential severity of such a complication.

Intrinsic obstetric causes include those due to the position of the mother during labour, such as prolonged hip flexion or increased lumbar lordosis, or direct compression by the fetal head on nerve structures such as the lumbosacral plexus. Instrumental delivery is associated with postnatal neuralgia due to maternal position and the risk of direct pressure from the forceps. Neurology secondary to regional anaesthesia could be caused by direct damage from the epidural needle, either to a nerve root or direct to the spinal cord, an epidural or spinal haematoma or abscess, arachnoiditis, meningitis or cauda equina syndrome.

How would you assess this patient?

I would review this patient's medical notes and drug chart, take a history and perform a full neurological examination. On review of medical notes, I would look particularly at the insertion of the epidural, were there any complications noted and whether there were multiple attempts. I would also look at the delivery; looking at whether it was a prolonged labour, any difficulties encountered and time in the lithotomy position. I would screen the patient's drug chart for anticoagulants, steroids or hypoglycaemics. When taking the history, I would focus on her sensory loss, the timing, pain, any motor symptoms and especially any deteriorating symptoms. I would ask about her previous history looking for factors that predispose to neuropathy, like backache, obesity or diabetes. I would carefully document all my findings from neurological examination, as any change in findings on further examination needs to be recognised.

After history and examination you find that this patient had a prolonged 2nd stage of labour, requiring forceps delivery in theatre. She has isolated sensory loss on the anterolateral aspect of her left thigh, with no other significant medical history or risk factors. What is the most likely cause of her neuropathy? How would you manage this case?

The pattern of sensory loss and history of delivery are consistent with injury to the lateral cutaneous nerve of the thigh, also known as meralgia paresthetica. This nerve can become compressed as it passes under the inguinal ligament, often from prolonged

lithotomy. The patient does not need any further investigation at this stage, but reassurance and careful documentation. I would suggest she is followed up by an anaesthetist daily prior to discharge. I would advise the patient that most cases resolve spontaneously within a few weeks, and a maximum of two months. If they do not resolve within this period she should seek medical advice. If there is any deterioration of symptoms, she should call the maternity unit urgently.

On your postnatal ward round you identify a woman who complains of a headache. She had an epidural for labour and an uneventful vaginal delivery 24 hours ago. What are the possible causes of her headache and how might you differentiate between them?

This question is testing your ability to produce a relevant differential diagnosis.

There are many causes for a postnatal headache. It is important to take a thorough history, examine the patient and order appropriate investigations in order to ascertain the possible diagnosis.

The headache might be due to a neurological condition, a generalised condition or secondary to an anaesthetic intervention. Neurological conditions include simple headache, migraine, meningitis, encephalitis, benign intracranial hypertension, cortical vein thrombosis, intracranial bleed and tumour. Generalised conditions include pre-eclampsia, dehydration and stress. With regards to anaesthetic intervention, the woman may have developed a post-dural puncture headache (PDPH).

What are the features of a PDPH?

The classical features of a PDPH include an onset within 72 hours of neuraxial blockade with a severe frontal or occipital headache. This is worse when sitting, moving suddenly, coughing and straining and is relieved when supine. Neck stiffness and photophobia may occur. Rarely cranial nerve palsies and even subdural or intracranial haemorrhage have been reported.

How would you treat a suspected post-dural puncture headache?

I would discuss the possible diagnosis with the woman and advise that she should try and remain well hydrated and take oral analgesia as tolerated. I would screen for some of the serological markers of sepsis by sending off bloods for a white cell count and CRP and would also monitor the patient's temperature. The patient may require laxatives to avoid straining during bowel motions. If the headache persisted I would consider performing an epidural blood patch.

When would you perform an epidural blood patch?

Performing a blood patch within the first 48 hours of the dural tap is associated with a higher failure rate of the procedure, so it is best to wait until then. If the patient is having severe, debilitating symptoms, it can be performed sooner. However, I would counsel the mother that there is a higher likelihood of failure, and that a repeat procedure may be required.

What is the success rate with an epidural blood patch?

Success rates from epidural blood patch have previously reported as being quite high, up to 90%; however, many women report a recurrence of symptoms. Women should be counselled that the chances of complete cure are 50%, and a second blood patch is required in 40% of cases.

How would you perform an epidural blood patch?

Pre-procedure I would ensure written consent and review the anaesthetic chart for when the last dose of LMWH was given.

The procedure requires two anaesthetists, one of whom should be a consultant, so I would have a consultant colleague to assist. Both of us would be scrubbed in and fully aseptic, with one assigned the role of taking blood, and the other performing the blood patch. The patient should be positioned in lateral position, as it may be uncomfortable for them to sit up. I would find the epidural space using a Touhy needle, using a loss of resistance technique and midline approach at the same level at which the dural puncture occurred, or within one space above or below. When the epidural space has been confirmed, the assisting anaesthetist will draw 20 ml of blood from a vein into a syringe and pass it to me. I would inject the blood slowly, stopping if the patient feels any significant discomfort.

Post-procedure, the patient should continue to lie flat for 2 hours and be reviewed by an anaesthetist every day until discharge.

You are the registrar on delivery suite and are called urgently to see a 28-year-old primiparous woman in labour at term, which had been progressing without complication. She had an epidural sited without complication by the anaesthetic SHO over 4 hours ago and had initial relief of pain following administration of 30 ml of 0.25% bupivacaine. She has been receiving regular bolus doses since, but has suddenly become agitated, and is experiencing facial and peripheral twitching. Her blood pressure is 170/105 mmHg, with a heart rate of 130 beats/min, and an irregular pulse. What is the likely cause of her sudden deterioration? How would you manage this situation?

The history and presenting features, altered mental state, agitation, and arrhythmia, strongly suggest local anaesthetic toxicity. Other causes should not be ruled out, and a team member should review this patient's notes and history for other conditions, such as epilepsy, heart disease or eclampsia while treatment is ongoing. Local anaesthetic toxicity is a risk in labour, due to possible misplacement or migration of an epidural catheter into a blood vessel and a reported increased sensitivity to local anaesthetic toxicity in pregnancy.

I would treat local anaesthetic toxicity by stopping any infusion of local anaesthetic and considering lipid emulsion treatment; a bolus injection of 1.5 ml/kg of 20% emulsion

given over 1 minute followed by an infusion of 15 ml/kg/hr. Further bolus doses can be given 5 minutes apart for treatment, up to a total of three bolus injections. I would continue ABCDE supportive therapy, while applying cardiac monitoring and assessing the effects of my treatment. If cardiac monitoring shows cardiac arrhythmia, I would treat this conventionally, being aware it may be resistant to treatment. There is a risk of seizure given her neurological symptoms, so I would ask for lorazepam, as well as preparing for anaesthesia and intubation.

This is a dynamic situation, requiring involvement of multiple team members; fetal monitoring should be obtained and emergency caesarean section should be considered at all times.

In the event of a cardiac arrest in a term mother, how should cardiopulmonary resuscitation be performed?

Cardiac arrest if confirmed, should prompt immediate initiation of CPR and escalation to the most senior obstetric, anaesthetic and neonatal staff. The physiological changes of pregnancy and the presence of the fetus necessitate some key changes to the ALS algorithm, namely that resuscitation should occur with 30° lateral tilt; we should aim for early endotracheal intubation and perimortem C-section should be immediately performed if there is no return of circulation within 4 minutes. Continued resuscitation should be according to the standard ALS algorithm.

If left lateral tilt cannot be immediately achieved, a team member should manually displace the uterus. Early intubation is indicated because of the higher risk of gastric aspiration in these patients. Pregnancy and peri-arrest scenarios both increase the likelihood of failed intubation, so video laryngoscope, if available, would be first line, and the first attempt to be made by the most senior anaesthetist present. A supraglottic airway should be placed in the event of failed intubation. Large bore IV access should be obtained above the level of the diaphragm. Effective CPR achieves 30% of normal cardiac output, and the uterus takes about 30% of cardiac output at term, thus CPR is insufficient to sustain life in mother and baby. The decision for peri-mortem C-section should be taken after 2–3 minutes if there has been no return of circulation. This does not need to occur in theatre and should be started within 4 minutes of initiating CPR, completing by 5 minutes.

Ongoing resuscitation should follow the ALS algorithm, considering all the possible causes of the 4 Hs and 4 Ts. In pregnancy, we must remember key causes such as amniotic fluid embolism, massive haemorrhage, eclampsia or magnesium toxicity. Non-obstetric causes that are associated with pregnancy include pulmonary embolism, sepsis and cardiac disease.

The MBRRACE report in 2021 looked at cases of mortality in 2017–2019. What can you tell me about the causes of obstetric mortality?

The MBRRACE report is published regularly so this discussion may now be out of date. Please read the most up to date version.

Maternal deaths are classified as direct or indirect; direct deaths are those related to an obstetric complication or resulting from any obstetric treatment. Indirect deaths are associated with an illness, which may be exacerbated by pregnancy or labour. Death from

any cause occurs at a rate of 8.79 per 100,000 maternities. The three most common direct causes of death are thrombosis and thromboembolism, obstetric haemorrhage and pregnancy-related sepsis.

There was one death as a result of anaesthesia over this period. Recommendations with greatest relevance to anaesthetic practice were on the management of maternal haemorrhage, the management of those receiving anticoagulation, and care of women in recovery.

The most common causes of indirect death are cardiac disease and neurological disease.

Did the MBRRACE report identify any patient characteristics associated with maternal mortality?

There were socioeconomic, demographic and medical characteristics associated with higher mortality rates. Women at severe social disadvantage made up 8% of maternal deaths, the three biggest elements of disadvantage being, mental health problems, substance abuse and domestic abuse. Women from Black and minority ethnic groups demonstrate a 2–4 times higher mortality rate when compared to the white population. Mortality rates were also higher for women less than 20 years of age or higher than 40. Of maternal mortalities, 65% were known to have a pre-existing medical problem, and 23% were obese.

Further Reading

Knight M, Bunch K, Tuffnell D et al. Saving lives, Improving mothers' care: Lessons learned to inform maternity care from the UK and Ireland Confidential Enquiries into Maternal Deaths and Morbidity 2017–19.

Mothers and Babies: Reducing Risk through Audits and Confidential Enquiries across the UK: MBRRACE-UK.

Madden A-M, Meng M-L. Cardiopulmonary resuscitation in the pregnant patient. *BJA Education*. 2020; 20(8): 252–258.

2.6.4 Pre-eclampsia – Jade A Loughran and Sarah F Bell

Try to keep your answer relevant to the particular case presented to gain maximum marks. You are asked to review a 28-year-old primigravida who is 36 weeks pregnant. She has been recently admitted to the labour ward for an induction of labour and is known to have hypertension.

What are the important issues that you would like to explore in her history?

The examiner is looking to see that you have had plenty of obstetric experience and that you can imagine yourself in this situation. A structured approach is vital.

When assessing this patient I would want to discuss both her obstetric history and her past medical and anaesthetic history. I would ask about why she is being induced and the history of this pregnancy and any previous pregnancies. I would ask about her past medical history, anaesthetic history, allergies and drug history. With regards to the blood

pressure, I would like to know what the recent readings have been, what treatment she is on and how long she has been taking any treatments, and whether she had hypertension prior to pregnancy. I would also ask about any symptoms of pre-eclampsia.

How is hypertension in pregnancy classified?

Hypertension associated with pregnancy is usually defined as a systolic blood pressure of greater than 140 mmHg, or a diastolic blood pressure of greater than 90 mmHg. Hypertension in pregnancy may be chronic hypertension, gestational hypertension or pre-eclampsia.

Chronic hypertension is that which exists before pregnancy or is detected before 20 weeks gestation. New hypertension after 20 weeks gestation, without significant proteinuria defines gestational hypertension, and pre-eclampsia is hypertension after 20 weeks gestation with proteinuria. This may be a urinary protein:creatinine ratio of greater than 30 mg/mmol, or a 24-hour urine collection with greater than 300 mg/L protein.

How common are hypertensive disorders in pregnancy?

Pre-eclampsia occurs in approximately 2–3% of pregnancies. Other hypertensive disorders are more common, with gestational hypertension occurring in approximately 6% of pregnancies, and chronic hypertension affecting up to 5% of pregnancies.

How do you diagnose pre-eclampsia?

Pre-eclampsia is a multisystem disease. The main features are the triad of hypertension, oedema and proteinuria. The hypertension is diagnosed as a systolic greater than 140 mmHg, diastolic greater than 90 mmHg or a mean blood pressure greater than 105 mmHg. Alternatively it is described as an increase in systolic or diastolic pressure of more than 30 mmHg or 15 mmHg respectively. The proteinuria is greater than 0.3 g/L in 24 hours.

Why does pre-eclampsia develop?

Try not to get too bogged down in this answer since it is complicated and not yet fully understood.

The exact cause of pre-eclampsia is unknown and there may be a genetic susceptibility. Impaired trophoblastic invasion leads to failure of the normal vasodilatation of blood vessels within the placental bed, leading to placental hypoperfusion and hypoxia. This causes the release of cytokines and inflammatory markers which leads to widespread endothelial dysfunction including altered capillary permeability and abnormal activity of the coagulation cascade, leading to multiorgan effects.

What are the features of pre-eclampsia?

Try and use your systems approach to keep structure to your answer.

The symptoms and signs of pre-eclampsia may be split into the different organ systems. In the cardiovascular system the systemic vascular resistance is raised and hypertension is a feature. The plasma volume is reduced when compared to the expansion normally seen in pregnancy. Changes in capillary permeability and reduced plasma oncotic pressure lead to peripheral oedema. Furthermore, the combination of raised

pulmonary artery pressure, low plasma oncotic pressure and altered capillary permeability in the lungs make these patients particularly susceptible to pulmonary oedema. With regards to the kidneys, a reduction in renal blood flow, glomerular filtration rate and urine output may be observed. Renal failure may develop. The haematological system may be affected with a fall in fibrinogen and platelet levels as seen in patients with HELLP syndrome (haemolysis, elevated liver enzymes and low platelets). The gastrointestinal system may be involved as part of HELLP syndrome and the patient may experience abdominal pain due to oedema and enlargement of the liver capsule. Finally, with regards to the neurological system the patient may describe visual disturbance and headaches. Hyperexcitability, hyperreflexia and papilloedema may be demonstrated and the patient may develop seizures.

How might pre-eclampsia affect the fetus?

The reduced placental perfusion may be compounded by ischaemia and infarction. This can lead to intrauterine growth restriction. The incidence of placental abruption and preterm labour is also increased.

Are there any risk factors for pre-eclampsia?

Yes, these can be patient or pregnancy-related factors. General risk factors include a family history of pre-eclampsia, maternal age over 40, BMI greater than 35 kg/m^2 , or pre-existing diabetes, hypertension, chronic kidney disease or antiphospholipid syndrome. Pregnancy-related risk factors include nulliparity or inter-pregnancy gap of more than 10 years, pre-eclampsia in a previous pregnancy, and multiple pregnancy.

What is the treatment for pre-eclampsia?

There is no specific answer to this question. The examiners are looking to see that you have an understanding of the general issues involved.

The treatment for pre-eclampsia is delivery of the baby, but this must be balanced against the gestational age and the potential risks associated with uncontrolled hypertension. In many cases antihypertensive treatment will be commenced in order to regain control of blood pressure prior to delivery; but this does not prevent the disease progression. Close monitoring of observational parameters, haematological and neurological indices are indicated in these patients both before and after delivery.

What antihypertensive treatment might this woman be taking?

Try and think back to a case you have seen.

Pharmacological treatment should be offered to any woman with blood pressure over 140/90 mmHg, aiming for 135/85 mmHg or lower. Oral labetalol is often a first-choice agent, but patients may also be treated with nifedipine, hydralazine or methyldopa.

What is the role of magnesium in pre-eclampsia and eclampsia?

Can you tell me anything about the MAGPIE trial?

The Collaborative Eclampsia Trial was a large international study evaluating the effects of magnesium sulphate given to women with pre-eclampsia. Magnesium sulphate more than

halved the risk of eclampsia. Currently magnesium sulphate is used to treat pre- and postpartum seizure activity. A loading dose of 4 g is given over 10 minutes followed by an infusion of 1 g/hour. Recurrent seizures are treated with further 2 g boluses of magnesium.

What clinical assessments would you perform on this woman and what would you be looking for?

I would examine the cardiorespiratory, neurological and gastrointestinal systems looking for any signs of pre-eclampsia. These might include hypertension, peripheral oedema, respiratory compromise, hyperreflexia, clonus, abdominal discomfort and right upper quadrant tenderness. I would also assess the patient's airway looking for factors which might predispose to a difficult intubation.

What would you consider necessary investigations?

Urine dipstick will indicate proteinuria. Urinary protein:creatinine ratios are often used, or 24-hour urine collection could be used to quantify this further.

I would request the results of a full blood count, urea and electrolytes, liver function tests, coagulation studies and ensure that a sample of blood had been sent for a group and save. I would be looking for anaemia, thrombocytopenia, renal failure, hepatic derangement and clotting abnormalities.

What advice would you give this woman regarding an epidural?

This question is assessing whether you have appreciated the role of epidural analgesia in women with pre-eclampsia.

A good working epidural placed early during labour has the potential to reduce the catecholamine release associated with pain. This should improve placental blood flow, thus benefiting both mother and fetus. It may also be rapidly topped up for instrumental delivery or emergency caesarean section. I would discuss these benefits with the patient; but also explain the risks of epidural placement. I would list the complications with the level of risk as published by the Obstetric Anaesthetic Association. These are: failure or patchy epidural (1 in 8 women need additional pain relief), nausea and vomiting, hypotension (1 in 50 women), headache (1 in 100 women), nerve damage (1 in 1000 for temporary nerve damage and 1 in 13000 for permanent nerve damage), and an increased risk of instrumental delivery.

What haematological parameters would you consider it safe for performing an epidural in this case?

I would need to review a full blood count and a clotting screen prior to the insertion of a spinal or epidural needle. I would consider a platelet count below $100 \times 10^9/L$ a contraindication for an epidural. An abnormal clotting screen with platelet counts above $100 \times 10^9/L$ would also be of concern. I would also review the white cell count. A leucocytosis of $25 \times 10^9/L$ or more can be normal in labour. If the white cell count were raised I would check the patient's temperature and look for a source of sepsis prior to considering inserting an epidural. The risk of epidural infection needs to be weighed against the potential benefits of epidural placement and discussed with the patient.

What about the platelet count considered safe for performing a spinal?

In my department the criteria are slightly different for spinal anaesthesia and a platelet count above $70 \times 10^9/L$ with normal clotting screen is acceptable for a spinal anaesthetic.

How would you manage this patient's fluid balance?

Try and think of what your unit's protocol says!

Management of fluid balance in patients with pre-eclampsia can be extremely challenging. It is important to carefully monitor the input and output, usually with a urinary catheter in situ. Renal function blood tests should be regularly checked. Oliguria is common in pre-eclampsia but does not necessarily imply volume depletion. Overhydration may contribute to the development of pulmonary oedema, but overenthusiastic fluid restriction or additional blood loss may also lead to renal failure.

In my unit a fluid restriction regime of 80 ml/hour is generally adopted, to include oral fluids, medications and IV fluids, with a target urine output of 0.5 ml/kg/hr. If the urine output falls then 250 ml judicious fluid boluses are considered, up to 500 ml. If this fails, 20–60 mg frusemide may be given.

Once the epidural is sited and the test dose has been administered the patient has a tonic–clonic seizure. What are the possible causes for this?

The seizure may be due to an obstetric or non-obstetric condition. This classification can be further split into neurological, non-neurological or anaesthetic-related causes.

Neurological obstetric conditions might include eclampsia, subarachnoid haemorrhage or intracranial haemorrhage. Non-obstetric neurological conditions such as pre-existing epilepsy, intracranial mass or infection may have precipitated the seizure.

Respiratory conditions that cause significant hypoxia or hypercapnia can initiate seizures. These might be related to the pregnancy such as amniotic fluid embolism or pulmonary embolism or be due to pre-existing conditions.

Metabolic conditions causing seizures include hypoglycaemia and hyponatraemia. These may be related to the pregnancy (for example a gestational diabetic who is starved and has received insulin) or due to a pre-existing condition.

Finally, with regards to anaesthetic interventions, the epidural may in fact be intrathecal leading to a high spinal or the epidural solution may have been injected into a vein leading to local anaesthetic toxicity.

What would be your initial management of this scenario?

Try and picture yourself in this situation and describe how you would manage it.

This is an obstetric medical emergency that needs prompt management. I would delegate a member of staff to call the labour ward resuscitation team including senior anaesthetic help and an ODP. I would also request the emergency eclampsia box and have this available. I would start by assessing the airway, give 100% oxygen via a face mask and ensure that the patient was in left lateral tilt. I would then quickly examine the respiratory system and check the oxygen saturation level. The blood pressure, heart rate and any obvious blood loss would need to be measured. I would obtain IV access and send bloods for FBC, U&E, LFT, glucose, coagulation studies and cross-match if not

already done. The obstetric team should examine the abdomen and uterus. I would give a bolus of 4 g magnesium sulphate IV in an attempt to halt the seizure. Depending on whether the seizure stopped and the mother regained consciousness, I would consider intubating the mother at this stage. Further pharmacological treatment with antihypertensive drugs, magnesium sulphate infusion and benzodiazepines may be required. The baby may need to be delivered by emergency caesarean section. A full neurological assessment should be performed as soon as possible and as a minimum the pupils should be assessed during the emergency.

The fit subsides and the woman partially regains consciousness, but the obstetricians are keen to proceed to a category 1 emergency caesarean section due to fetal distress. There is not time to top up the epidural and you are required to perform a general anaesthetic. How would you perform this?

Having informed my senior support of the situation and asked for their assistance, I would prepare for a general anaesthetic while also ensuring that appropriate maternal and fetal resuscitation were ongoing. I would ensure that I had at least one skilled assistant, a tilting table, suction and a checked anaesthetic machine. I would administer 30 ml 0.3 M sodium citrate if the patient was awake enough to swallow. I would preoxygenate to achieve an end-tidal oxygen level of 93% (or for 3 minutes) in a left lateral tilt position. During preoxygenation I would expect the obstetricians to be scrubbed and preparing the abdomen so that they are ready to start. I would prefer to have an arterial line in situ to closely monitor blood pressure but would not delay the induction of anaesthesia unnecessarily. I would induce anaesthesia using a rapid sequence induction technique including a short-acting opiate to obtund the pressor response to laryngoscopy. This would need to be communicated to the attending neonatologist. I would secure the airway with an endotracheal tube and confirm correct positioning prior to allowing the commencement of surgery. I would maintain anaesthesia using a 50:50 nitrous oxygen mix and sevoflurane. I would aim to achieve a mild respiratory alkalosis to maintain this physiologically normal adaptation of pregnancy. After delivery I would give syntocinon 5 international units as a slow bolus. During the operation I would administer cautious fluid therapy and continue standard AAGBI monitoring including temperature, BIS and peripheral neuromuscular monitoring.

With regards to postoperative analgesia, I would consider regional nerve blocks such as bilateral ilioinguinal nerve blocks, transversus abdominis or quadratus lumborum blocks, or use the epidural to administer morphine and fentanyl. I would prescribe regular paracetamol and consider a non-steroidal anti-inflammatory such as diclofenac; although in this particular case I would not give this drug due to the risk of worsening the patient's renal function.

What potential problems might you anticipate during and after the operation?

The problems may be divided into cardiovascular and haematological, respiratory, neurological and renal. Cardiovascular instability may occur at induction, laryngoscopy

and intubation, intra- or post-op. Dramatic swings in blood pressure may require prompt treatment. Hypertensive crises may lead to intracranial haemorrhage and bleeding at the operative site. This may be complicated further by coagulopathy. Fluid balance should be carefully monitored with restricted intake and close observation of urine output. With regards to the respiratory system, the intubation may be difficult due to airway oedema and the patient may be difficult to ventilate due to pulmonary oedema.

Postoperatively the patient will require obstetric high dependency care. She will require ongoing magnesium infusions, and possibly antihypertensive medications. She may have further seizures and require admission to a critical care unit for respiratory, neurological, haematological or renal support.

Further Reading

British National Formulary 59. 2010.

Leslie D, Collins RE. Hypertension in pregnancy. *BJA Education*. 2016; 16 (1):33–37.

NICE. Hypertension in pregnancy: diagnosis and management (NG133).

OAA. Pain Relief in Labour 3rd Edition. 2012.

2.6.5 Labour Analgesia – Shakira Nathoo

Can you describe the pain pathways during the first and second stages of labour?

The pathways can be divided into visceral and somatic. During the first stage of labour, pain is primarily visceral and is caused by cervical dilation and uterine contraction. The afferent nociceptors are unmyelinated C fibres. They accompany sympathetic fibres through cervical, uterine and hypogastric nerve plexuses into the sympathetic chain and enter the spinal cord at T10–L1.

Pain during the second stage is primarily somatic due to stretching of the vagina and perineum, as well as any tissue damage that may occur. The afferent nociceptors are A-delta fibres. Transmission is via the pudendal nerve which enters the spinal cord at S2–4.

All nerve fibres pass via the dorsal horn and ascend via the spino-thalamic tract to the brain.

How can the parturient manage pain during labour?

Pain management during labour can be divided into non-pharmacological, pharmacological and regional analgesia.

There are a wide range of non-pharmacological methods which include continuous support from a care-giver, immersion in water, massage, acupuncture, TENS, sterile water injections, aromatherapy, breathing techniques, relaxation techniques and hypnotherapy.

Commonly available pharmacological agents are paracetamol, codeine, inhaled nitrous oxide, pethidine and remifentanyl. Other intravenous and intramuscular opiates have been used but this is not common practice in the UK.

What is pethidine? Describe the dosing and route of administration used for labour analgesia.

Pethidine is meperidine, a synthetic phenylpiperidine derivative. It is metabolised to the active form normeperidine. It is commonly administered as an intramuscular injection at a dose of 1 mg/kg with a maximum dose of 150 mg.

What are the side effects of pethidine?

There are side effects for the mother and the fetus.

Maternal side effects are similar to other opioids. They include confusion, sedation, delayed gastric emptying, hypoventilation and dose dependent respiratory depression. Normeperidine has convulsant properties and is contraindicated in pregnancy-induced hypertension.

Meperidine is highly lipid-soluble and readily crosses the placenta. Fetal plasma concentration peaks 2–3 hours following maternal administration and meperidine accumulates in the fetus, therefore fetal side effects are dependent on dosing and timing of administration. The primary side effect is respiratory depression leading to lower APGAR scores. The newborn is at risk of being less alert and less successful in initially establishing breastfeeding.

Explain the key pharmacokinetics and pharmacodynamics of remifentanyl that are relevant in its use for labour analgesia.

Remifentanyl is an ultrashort-acting opioid. It has a rapid onset time of 1 minute and its context sensitive half time is 3 minutes. It is degraded by plasma and nonspecific tissue esterases. These characteristics allow it to be timed with a contraction. Remifentanyl is highly lipophilic therefore there are high rates of placental transfer. It is, however, rapidly redistributed and metabolised in the fetus.

Can you tell me about the safety precautions that should be taken when using a remifentanyl PCA?

Remifentanyl PCA for labour should only be used in a setting with the appropriate guidelines, equipment and training of staff.

There must be direct one to one midwifery care throughout its use. If the midwife needs to step out of the room, the PCA must be discontinued during this time.

Equipment must include a lockable reservoir of drug, a pump with predetermined programmes and dedicated giving set. The remifentanyl PCA must have a dedicated peripheral venous cannula which should not be flushed once the PCA is running.

The anaesthetist should set up the PCA and be present for the first doses administered.

Monitoring must include continuous oxygen saturations, respiratory rate, pain score and sedation score and should be documented appropriately initially every 15 minutes and then every 30 minutes. Monitoring should continue for 30 minutes after the PCA has been discontinued.

Nasal specs for oxygen delivery should be immediately available and oxygen should be commenced if oxygen saturations fall below 95%.

Naloxone must be prescribed and readily available.

The parturient should understand how to use the PCA. They must press the PCA button at the onset of contraction. Pressing the button after this increases the risk of respiratory depression and does not provide optimum analgesia. Everyone involved in their care, including the birth partners, must understand that the parturient is the only person permitted to press the PCA button.

What is a typical dose of remifentanyl?

40 mcg bolus with a 2 minute lockout. Then can be reduced if the parturient has signs of respiratory depression despite correct usage.

Are you aware of any evidence base which supports the use of remifentanyl PCAs?

The RESPITE study was a multicentre, randomised control trial comparing the rate of conversion to epidural between intramuscular pethidine and remifentanyl PCA. The rate of conversion to epidural was double in the pethidine group (41% vs 19%). There were more vaginal births and a lower rate of instrumental deliveries in the remifentanyl group.

You are asked to review a low risk primiparous woman who had an epidural sited for labour analgesia eight hours ago. The midwife is concerned as she has a Bromage score of 1 and is profoundly insensate. Can you discuss your assessment and any actions you would take?

I would promptly assess the woman. I would ask about the progression of her sensory and motor blocks and whether these have become denser over time. I would establish the relationship between epidural doses given and her symptoms. I would establish the height of the block and perform a neurological examination of the lower limbs. I would also review any medical history that she has and review any relevant investigations.

I would examine the anaesthetic chart for any issues or complications there were in siting the epidural. I would interrogate the PCA pump to see how often the woman is pressing her PCA button and see whether she has had any additional top ups. I would aspirate the epidural catheter to look for signs of intrathecal migration.

Depending on my findings, actions that I may consider may be discontinuing the epidural infusion and monitoring, stopping the PCA if the catheter is intrathecal and moving to anaesthetist-only top-ups, or removing or resiting the epidural. If the woman has risk factors for epidural haematoma or abscess or if there is no neurological improvement despite stopping the PCA, she is likely to need imaging and neurosurgical referral. Regardless, I would discuss this patient with my consultant and keep her under review.

Can you explain to me how your assessment and management would differ if this woman was 4 hours postpartum after an uneventful normal vaginal delivery and cessation of epidural and could still not perform a straight leg raise?

I would perform the same assessment as the previous woman. I would enquire about symptoms of fever, localised back pain, or radiating or lancinating pain suggestive of

radiculopathy. Crucially, if there is continued resolution of sensory and motor block this is reassuring and the woman should have ongoing neurological monitoring and be kept under review. If there is sustained or progressive sensory and motor blockade despite not receiving any further epidural analgesia, she should undergo urgent imaging and neuro-surgical referral. Again, I would discuss this with my consultant.

Further Reading

- Alleemudder DI, Kuponiya Y, Kuponiya C, McGlennan A, Fountain S, Kasivisvanathan R. Analgesia for labour: An evidence-based insight for the obstetrician. *The Obstetrician and Gynaecologist*. 2015;17:147–55.
- Cambic CR, Wong CA. Labour analgesia and obstetric outcomes. *British Journal of Anaesthesia*. 2010;105(1):50–60.
- Fortescue C, Wee MY. Analgesia in labour: Non-regional techniques. *BJA Education*. 2005;5(1):9–13.
- Labor S, Maguire S. The Pain of labour. *Reviews in Pain*. 2008;2(2):15–19.
- Obstetrics Anaesthetists' Association. Remifentanyl [Internet]. 2018 [cited 18 July 2021]. Available from: www.oaa-anaes.ac.uk/ui/content/content.aspx?ID=68
- Ronel I, Weiniger CF. Non-regional analgesia for labour: Remifentanyl in obstetrics. *BJA Education*. 2019;19(11):357–361.
- Shatil B, Smiley R. Neuraxial analgesia for labour. *BJA Education*. 2020;20(3):96–102.
- Wilson MJA, MacArthur C, Hewitt CA, Handley K, Gao F, Beeson L, Daniels J. Intravenous remifentanyl patient-controlled analgesia versus intramuscular pethidine for pain relief in labour (RESPITE): An open-label, multicentre, randomised controlled trial. *The Lancet*. 2018;392:662–672.
- Yentis SM, Lucas DN, Brigante L, Collis R, Cowley P, Denning S, Fawcett WJ, Gibson A. Safety guideline: Neurological monitoring associated with obstetric neuraxial block 2020: A joint guideline by the Association of Anaesthetists and the Obstetric Anaesthetists' Association. *Anaesthesia*. 2020;75(5):913–919.

2.6.6 Placenta Praevia – Shakira Nathoo

Can you explain how placenta praevia is diagnosed and graded?

Placenta praevia is diagnosed by performing a transvaginal ultrasound scan after 20 weeks of pregnancy. It may present with painless antepartum haemorrhage or parturients may be asymptomatic. Placenta praevia is a condition where the placenta lies directly over the internal cervical os. A placenta which lies less than 20 mm from the os is defined as a low-lying placenta. Parturients with either placenta praevia or a low-lying placenta will undergo further ultrasound scans to monitor placental site and inform mode of delivery.

Placenta praevias can be subdivided into four grades. Grade 1 describes the lower edge of the placenta being inside the lower uterine segment. In grade 2 the lower edge reaches the internal os. In grade 3 the placenta partially covers the cervix and in grade 4 the placenta completely covers the cervix. The term 'minor' placenta praevia refers to grades 1 and 2 and the term 'major' refers to grades 3 and 4.

What are the risk factors for developing placenta praevia?

Risk factors include previous caesarean section, assisted reproductive technology, maternal smoking, advanced maternal age, previous placenta praevia, multiparity, previous uterine surgical procedures, polyhydramnios and birth intervals less than 1 year or over 4 years.

How can the spectrum of the abnormally invasive placenta be subdivided?

This spectrum includes placenta accreta where the placenta is directly adherent to the myometrium, placenta increta where the placental chorionic villi invade into the myometrium, and placenta percreta where the villi invade through the myometrium into the serosa and may invade into nearby organs.

You are asked to review a multiparous woman in the high-risk clinic prior to her elective caesarean section for placenta praevia. What are the key issues you would discuss with her?

In addition to taking a full history and performing an airway examination there are a number of key issues I would discuss. Firstly, I would ensure that the woman was aware of her high risk of bleeding and I would have expected her to have been counselled by the obstetric team prior to our consultation. I would want to ensure that she was willing to accept blood products and I would explore any concerns that she had surrounding this. I would ensure she had a recent haemoglobin result and ensure antenatal anaemia is investigated and treated in the available timeframe.

Secondly, I would discuss the conduct of anaesthesia. I would initially opt for a regional anaesthetic but counsel the woman of the risk of conversion to general anaesthetic. If the procedure was likely to be complex or difficult, I could consider the use of combined spinal and epidural anaesthesia.

Thirdly, I would discuss the need for two wide-bore intravenous access points. I would also discuss the need for invasive monitoring and access during the procedure if they were required. I would also discuss her postoperative destination and care which are likely to be in the obstetric high dependency unit, but may need to be escalated to an intensive care unit.

Are there any other blood conservation strategies you could offer to this woman?

Intraoperative cell salvage could be offered to this woman. In addition to the standard risks, in the pregnant population there is the additional risk of resus sensitisation which the woman must be counselled about. If a woman is rhesus negative, a postoperative Kleihauer should be performed, and potential resus sensitisation is managed by giving appropriately dosed anti-D.

Should intraoperative cell salvage be set up for all women undergoing caesarean section?

This question was asked by the SALVO trial in 2018. Given the modest reduction in donor blood transfusion against the cost of cell salvage and maternal risks, it was not deemed cost-effective for routine use in caesarean section. The OAA and AAGBI have not recommended its routine use, therefore it is largely used in high-risk parturients and ongoing obstetric haemorrhage.

What additional preparation would be required for this elective caesarean section?

I would want to ensure involvement of the relevant team members. The case should be managed by a consultant obstetrician and anaesthetist. If the delivery was preterm, the neonatal team should have been consulted and a cot should be available if required. I would ensure blood bank and haematology team had been liaised with and that cross-matched blood was immediately available. I would ensure the availability of an HDU bed.

I would also want to ensure the availability of some additional equipment including a fluid warmer with a blood giving set run through, a rapid transfusion device and cell salvage. I would also ensure I had additional infusion pumps available. I would ensure the immediate availability of arterial lines, central lines and vasoactive drugs.

Further Reading

- Brennan K. Placental pathology: A review of placenta previa, placental abruption and placenta accreta. *Obstetric Anaesthesia*. 2019;51.
- Jauniaux ERM, Alfirevic Z, Bhide AG, Belfort MA, Burton GJ, Collins SL, Dornan S, Jurkovic D, Kayem G, Kingdom J, Silver R, Sentilhes L, on behalf of the Royal College of Obstetricians and Gynaecologists. Placenta Praevia and Placenta Accreta: Diagnosis and Management. Green-top Guideline No. 27a. *British Journal of Obstetrics and Gynaecology*. 2018.
- Khan KS, Moore P, Wilson M, Hooper R, Allard S, Wrench I, Roberts T, McLoughlin C, Beresford L, Geoghegan J, Daniels J. A randomised controlled trial and economic evaluation of intraoperative cell salvage during Caesarean section in women at risk of haemorrhage: The SALVO (cell SALVage in Obstetrics) trial. *Health Technology Assessment (Winchester, England)*. 2018;22(2):1.
- Klein AA, Bailey CR, Charlton AJ, Evans E, Guckian-Fisher M, McCrossan R, Nimmo AF, Payne S, Shreeve K, Smith J, Torella F. Association of Anaesthetists guidelines: Cell salvage for peri-operative blood conservation 2018. *Anaesthesia*. 2018;73(9):1141–1150.
- Mavrides E, Allard S, Chandraran E, Collins P, Green L, Hunt BJ, Riris S, Thomson AJ, on behalf of the Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum haemorrhage. *British Journal of Obstetrics and Gynaecology*. 2016;124:106–149.
- Obstetrics Anaesthetists' Association. Cell Salvage [Internet]. 2009 [cited 18 July 2021]. Available from: www.oaa-anaes.ac.uk/ui/content/content.aspx?ID=74.
- Smith C, Shippam W, Brown J, Abir G. Intraoperative cell salvage in obstetrics. Obstetric anaesthesia. *World Federation of Societies of Anaesthesiologists*. 2018.

2.6.7 Grown-up Congenital Heart Disease (GUCHD) in Pregnancy and Labour – Eluned Fisher

Tell me about the risks that pregnancy poses in Grown-up Congenital Heart Disease.

Recent medical and surgical advances in treatments for congenital heart disease (CHD) have led to more than 90% of children born with CHD surviving into adulthood. Most surgical interventions, however, are not curative, potentially requiring reoperation. CHDs represent the most common cause of cardiac-related maternal morbidity. GUCHD carries

a high risk for both mother and fetus in pregnancy. Many of these risks are mediated by volume overload and haemodynamic changes during pregnancy. Complications include cardiac failure, thrombosis, pulmonary hypertension (PH), aortopathy, and unexpected sudden cardiac death. Arrhythmias may also occur with increased prevalence with more advanced age; largely attributable to surgical scarring. Some of these arrhythmias have a significant negative impact on the life expectancy of women with CHD.

How would you counsel a woman pre-pregnancy with GUCHD?

Preconception counselling is vital for patients with GUCHD in order to inform and plan for contraception and conception. Failure in contraception may prove fatal for some with severe CHD. Preconception counselling should be carried out early by an experienced multidisciplinary team including maternal–fetal medicine specialists and cardiologists with an interest in pregnancy and CHD. Despite surgical repair, many women with CHD are not entirely cured of their physiological changes and many have sequelae with implications for pregnancy. Thorough assessment by a cardiologist is recommended in early pregnancy if regular care has not been received prenatally. All counselling should include generalised recommendations for a healthy pregnancy such as preconception folic acid, smoking and alcohol cessation and maintaining a healthy diet and weight management. More specialised cardiac advice includes education on the maternal and fetal risks in pregnancy and labour. Opportunities to optimise comorbidities prior to pregnancy include blood pressure management and modification of teratogenic medications.

It is also important to manage psychosocial aspects during the preconception and the peripartum period. Pregnancy can be a highly emotive and anxiety-provoking time for many women and it is common for patients with GUCHD to worry about the impact their pregnancy may have on their own health as well as potential risks to their baby.

What pre-pregnancy assessments may be necessary in GUCH?

A detailed history should be taken including cardiac, surgical and obstetric history, as well as functional status. A thorough examination should follow prior to investigations including a chest X-ray, a 12-lead electrocardiogram (ECG) and echocardiography. Functional capacity may be formally assessed with cardiopulmonary exercise testing (CPX). This is useful to measure haemodynamic response in women with aortic stenosis (AS), inducible ischaemia in women with coronary disease and to assess ventricular function in those with cardiomyopathies or valve abnormalities. Cardiac MRI should be considered when echocardiography proves insufficient in the diagnosis of complex CHD. Cardiac catheterisation to measure pulmonary artery pressures and Holter monitoring may also be conducted. Blood tests should use normal pregnancy reference values. For example, blood troponin can detect myocardial ischaemia as it is not normally increased in pregnancy and a normal B-type natriuretic peptide level may help to exclude cardiac decompensation.

What risk-assessment tools can be used for these patients?

There are a number of risk-assessment tools to predict maternal cardiac complications during pregnancy including:

- World Health Organization (WHO) classification
- Cardiac Disease in Pregnancy (CARPREG) risk score
- Pregnancy and Congenital Heart Disease (ZAHARA) risk score.

Which CHD types should be counselled to avoid pregnancy?

- Pulmonary Hypertension (Eisenmenger syndrome)
- LV outflow tract obstruction (AS with a mean pressure gradient >50 mmHg)
- NYHA > class III or EF <35%
- Mechanical valves
- Cyanotic heart disease (PaO₂ <85%)
- Marfan Syndrome (AV diameter >40mm)
- Fontan circulation
- Arrhythmias with inducible haemodynamic compromise
- Kawasaki disease with coronary aneurysm

What risks are there to the fetus of a woman with GUCHD?

Fetal complications follow a spectrum, from intrauterine growth restriction to miscarriage and stillbirth, in addition to the risks of prematurity including retinal and lung complications. Of concern to many GUCHD mothers, the genetic transmission of CHD is likely 3–5%, with rates as high as 10% in left-sided outflow tract lesions such as AS. There are associated risks to the fetus from maternal complications. For example, arrhythmias may lead to significant hemodynamic compromise for both mother and baby. Moreover, many pharmacological agents used in the management of cardiac pathology during pregnancy have adverse effects for both.

What physiological changes in pregnancy are most important in GUCHD patients?

Haemodynamic changes in pregnancy account for the majority of morbidity in GUCHD patients during pregnancy and may lead to cardiac dysfunction or failure. There is a decreased systemic vascular resistance which may cause reduction in cardiac output and a low peripheral perfusion pressure leading to deterioration in maternal health. Even in normal pregnancy there is an increase in circulating volume, which can cause the left ventricular (LV) end-diastolic pressure to rise due to volume overload. This may lead to pulmonary artery hypertension and pulmonary oedema. Peripheral oedema may also appear due to elevated venous pressure. Pregnancy results in a hypercoagulable state, thus meticulous care must be given to women with increased risk of clots including mechanical valves or atrial fibrillation (AF) and those prone to developing deep vein thrombosis, including Fontan patients whose leg veins may have been occluded. Arrhythmias also may have significant impact due to impaired autonomic nerve activity, volume overload and scarring postoperatively as well as newly induced cardiac failure. Subsequent tachyarrhythmias may cause decompensation during pregnancy and labour.

How would you care for a GUCHD mother in pregnancy?

It is important these women have regular monitoring throughout pregnancy by an MDT including obstetricians, cardiologists, anaesthetists and midwives. The frequency of this monitoring should depend upon risk in comparison to a normal healthy individual. In women with moderate to severe CHD, two-weekly monitoring from 15 weeks

gestation and subsequent weekly monitoring from 25 weeks gestation is recommended. If compromise occurs, women may be hospitalised for bedrest and management from 20 weeks gestation. Normal physiological changes in pregnancy, particularly those of the cardiovascular and respiratory systems, may mimic cardiac decompensation, hence the need for regular and careful evaluation of cardiac function.

Patients predicted to develop heart failure during pregnancy should ideally be repaired prior to conception. For example, women who have significant arrhythmias as a result of the GUCHD may have ablation or pacemaker insertion before pregnancy. If this is not possible direct current (DC) cardioversion or catheter ablation is reported to be safe and effective during pregnancy. The indication for invasive surgery during pregnancy should be severe cardiac failure which is life-threatening to both mother and fetus. The greatest risk to the fetus is in early pregnancy, whereas the mother is at greatest risk in late pregnancy. Therefore, intervention should be performed after 14–18 weeks gestation whereby fetal organogenesis has occurred. Care should be taken to protect the fetus from unnecessary radiation. If cardiopulmonary bypass (CPB) is required there is an associated fetal mortality of 9–30%. Mortality of the mother is comparably lower but with a high associated morbidity, thus it is better to avoid cardiovascular surgery during pregnancy when possible.

How would you care for a GUCHD mother during labour and delivery?

These women should be managed in a tertiary centre. Continuous monitoring includes ECG, blood pressure and oxygen saturations as well as fetal monitoring via a cardiotocography (CTG). Throughout labour these women should be managed in the left lateral position, avoiding IVC and aortal compression by the gravid uterus. Normal vaginal delivery is generally preferred over caesarean section due to reduced risks for both mother and fetus. Fewer changes in blood volume and reduced haemorrhage occur, with average blood loss during vaginal delivery being approximately 400–500 ml and almost double that, 800–900 ml, during caesarean section. There is also reduced coagulation complications and fewer infections. Care should be taken to minimise cardiovascular stress during labour, including effective analgesia commonly in the form of an early epidural. Excessive maternal exertion should be avoided by providing assistance during the active second stage of labour. Management of the third stage of labour with oxytocin is given as a continuous intravenous infusion to minimise side effects such as peripheral vasodilation, tachycardia and fluid retention, other uterotonics should be used with caution if required.

In women with CHD, it is more common to observe cardiac failure after delivery as opposed to during pregnancy, due to rapid increase in venous return via the placenta and relief of pressure on the IVC, which may lead to fluid overload and decompensation. The return to normal haemodynamics and normal cardiac function also takes longer in women with GUCHD, up to 6 months postpartum, with a risk that full restoration to pre-pregnancy function may never occur.

Further Reading

Dalvi BV, Chaudhuri A, Kulkarni HL.

Therapeutic guidelines for congenital complete heart block presenting in

pregnancy. *Obstetrics and Gynecology*. 1992; 79(5 (Pt 2)):802–804.

Drenthen W, Boersma E, Balci A et al.

Predictors of pregnancy complications in women with congenital heart disease.

- European Heart Journal*. 2010; 31 (17):2124–2132.
- Drenthen W, Pieper PG, Roos-Hesselink JW et al. Outcome of pregnancy in women with congenital heart disease: A literature review. *Journal of the American College of Cardiology*. 2007; 49(24):2303–2311.
- Niwa K, Akagi T, Aomi S. Guidelines for Indication and Management of Pregnancy and Delivery in Women with Heart Disease (JCS 2010): digest version. *Japanese Circulation Society Joint Working Group. Circulation Journal*. 2012; 76(1):240–260.
- Joglar JA, Page RL. Antiarrhythmic drugs in pregnancy. *Current Opinion in Cardiology*. 2001; 16(1):40–45.
- Johnson M, von Klemperer K. Cardiovascular Changes in Normal Pregnancy. *Heart Disease and Pregnancy*. 2nd ed. Cambridge: Cambridge University Press; 2016; 19–28.
- Kamiya CA, Iwamiya T, Neki R. Outcome of pregnancy and effects on the right heart in women with repaired tetralogy of fallot. *Circulation Journal*. 2012; 76(4):957–63.
- Kamiya C, Nakatani S, Hashimoto S et al. Role of echocardiography in assessing pregnant women with and without heart disease. *Journal of Echocardiography*. 2008; 6:29–38.
- Niwa K. Adult Congenital Heart Disease with Pregnancy. *Korean Circulation Journal*. 2018; 48(4):251–276.
- Niwa K, Tateno S, Akagi T et al. Arrhythmia and reduced heart rate variability during pregnancy in women with congenital heart disease and previous reparative surgery. *International Journal of Cardiology*. 2007; 122(2):143–148.
- Ohuchi H, Tanabe Y, Kamiya C, Noritake K et al. Cardiopulmonary variables during exercise predict pregnancy outcome in women with congenital heart disease. *Circulation Journal*. 2013; 77(2):470–476.
- Regitz-Zagrosek V, Blomstrom Lundqvist C, Borghi C et al. ESC Guidelines on the management of cardiovascular diseases during pregnancy: The task force on the management of cardiovascular diseases during pregnancy of the European Society of Cardiology (ESC). European Society of Gynecology (ESG), Association for European Paediatric Cardiology (AEPC), German Society for Gender Medicine (DGesGM). *European Heart Journal*. 2011; 32(24):3147–3197.
- Silversides CK, Kiess M, Beauchesne L et al. Canadian Cardiovascular Society 2009 Consensus Conference on the management of adults with congenital heart disease: Outflow tract obstruction, coarctation of the aorta, tetralogy of Fallot, Ebstein anomaly and Marfan's syndrome. *Canadian Journal of Cardiology*. 2010; 26 (3):e80–97.
- Siu SC, Colman JM, Sorensen S et al. Adverse neonatal and cardiac outcomes are more common in pregnant women with cardiac disease. *Circulation*. 2002; 105 (18):2179–2184.
- Siu SC, Sermer M, Harrison DA et al. Risk and predictors for pregnancy-related complications in women with heart disease. *Circulation*. 1997; 96(9):2789–2794.
- Steer PJ. Pregnancy and contraception. *Adult Congenital Heart Disease: A Practical Guide*. Oxford: Blackwell Publishing Ltd.; 2005; 16–35.
- Tanous D, Siu SC, Mason J et al. B-type natriuretic peptide in pregnant women with heart disease. *Journal of the American College of Cardiology*. 2010; 56 (15):1247–1253.
- Thorne S, MacGregor A, Nelson-Piercy C. Risks of contraception and pregnancy in heart disease. *Heart*. 2006; 92 (10):1520–1525.

2.6.8 Amniotic Fluid Embolism and Postpartum Haemorrhage (PPH) – Farzad Saadat and Sarah F Bell

PPH is both common and a potentially grave clinical scenario. You will be expected to demonstrate in-depth clinical knowledge and show clear thinking for a situation involving multiple team members and moving parts. While amniotic fluid embolism (AFE) is less

common, it is a potentially catastrophic complication, for which you should also show a good understanding.

You are the anaesthetist present at an elective caesarean section. You are looking after a healthy primiparous woman diagnosed with a breech presentation having her first caesarean section. She had an uneventful spinal anaesthetic and delivery of a live baby. The obstetricians are suturing the uterus when she becomes acutely short of breath. What potential differential diagnoses would you consider in this case?

This is a clinical question that is initially testing your ability to cover all the potential problems. Use a structured approach to try and remember as many possibilities as you can, while remember the important conditions particular to this situation!

There are a number of potential causes of dyspnoea in a woman undergoing an elective caesarean section under spinal anaesthesia. These may be divided into respiratory, cardiovascular or drug-related problems. The respiratory complication may be due to bronchospasm. This may be associated with asthma, hyperreactive airways in a smoker, anaphylaxis or aspiration. Further potential respiratory causes are hypoxia secondary to atelectasis, pulmonary oedema, pulmonary embolism (which might be amniotic fluid or blood clot) or even a pneumothorax. Cardiovascular complications such as acute haemorrhage or decompensation from pre-existing valve or cardiac disease may have caused the dyspnoea. Finally, there may be a drug-related problem such as a high spinal, anaphylaxis or drug reactions such as carboprost-induced bronchospasm.

How common is amniotic fluid embolism?

Amniotic fluid embolism (AFE) is uncommon. Its incidence varies from 1 in 8,000 to 1 in 80,000 pregnancies. It accounts for 0.32 maternal deaths per 100,000 deliveries. The disease process occurs most frequently during labour, or within 6 hours of delivery. Factors associated with an increased risk of developing AFE may be divided into maternal, fetal and uterine or placental. Maternal factors include multiparous mothers, older mothers and women with a history of atopy. Fetal factors include intrauterine death, meconium-stained liquor and microsomia. Uterine or placental factors include polyhydramnios, chorioamnionitis, strong or tetanic uterine contractions, uterine rupture and placenta accreta.

Can you describe the pathophysiology of AFE?

AFE is a complex condition with cardiovascular, respiratory and coagulopathy changes that result from a pathological maternal immune response. Amniotic fluid enters the maternal circulation (via ruptured membranes or uterine vessels) down a pressure gradient. The fetal antigens in the fluid stimulate the release of a cascade of biochemical mediators. Initially these cause pulmonary artery vasospasm followed by acute elevation of right ventricular pressures and right ventricular dysfunction. This in turn leads to hypoxaemia, hypotension and myocardial damage. The patient may consequently develop left ventricular failure, pulmonary oedema and an ARDS picture. Coagulopathy is also a feature of this condition.

What are the features of AFE?

The symptoms of AFE range from dyspnoea, cough and wheeze to headache and chest pain. Signs include fetal distress, hypoxia, tachypnoea, bronchospasm, hypocapnia (if intubated due to reduced cardiac output), tachycardia, hypotension, seizures, uterine atony, haemorrhage and coagulopathy.

Is there any specific management for suspected AFE?

No. The management is supportive and preventative. Resuscitation of the pregnant woman should always be performed in the left lateral position until the baby is delivered, with aggressive treatment of hypoxia, cardiovascular failure and coagulopathy.

How do you define maternal obstetric haemorrhage?

Maternal haemorrhage can be defined as antepartum, APH, or postpartum, PPH. Antepartum is any bleed after 24 weeks gestation and before delivery. Any APH over 50 ml or with signs of shock is considered significant.

PPH can be categorised as primary or secondary. Primary is any PPH within 24 hours of delivery, and secondary is between 24 hours and 12 weeks after delivery. The RCOG defines PPH as major if over 1,000 ml with ongoing bleeding and or clinical shock.

What is the blood flow to the uterus and why is this important?

Uterine blood flow increases from <5% to 10% of cardiac output at term, or 1 l/min. Thus, haemorrhage may be brisk and potentially fatal. In addition, the spiral artery remodelling that occurs during placental implantation means that these vessels lack a muscular endothelium. Therefore, after birth, mechanical contraction of the uterine muscle is required to compress these vessels and stop bleeding from the placental bed.

What clinical signs may be evident in a woman who is bleeding postpartum?

The respiratory rate will increase initially, and then a tachycardia will develop. This may be the only sign until 30–40% blood loss since maternal blood volume expands prior to delivery. Aortocaval compression will compound hemodynamic instability. Hypotension, reduced urine output and changes in conscious level will occur if bleeding continues.

What might be the first sign in a woman with antepartum haemorrhage?

The bleeding may be concealed and so fetal distress may be first sign.

What are the causes of obstetric haemorrhage?

The causes vary depending on the timing of the bleeding. Early pregnancy bleeding may be due to incomplete or septic abortion or a ruptured ectopic. Antepartum haemorrhage may be due to placenta praevia, abruption, uterine rupture or trauma.

Can you tell me any risk factors for abruption?

Yes, maternal hypertension, uterine over distension, previous abruption, smoking and trauma.

And what about uterine rupture?

Previous caesarean section is the most common risk factor.

Tell me about the causes of postpartum haemorrhage.

Postpartum haemorrhage is most commonly due to uterine atony. Additional causes include retained products, genital tract trauma, surgical trauma, abnormally adherent placenta, clotting defects or uterine inversion.

So what are the risk factors for uterine atony?

These include prolonged or augmented labour, uterine over distension, abnormalities or multiple gestation, placenta praevia, increased parity and increased maternal age.

And why might a pregnant or postpartum woman develop a clotting abnormality?

Well, most coagulation factors usually increase during pregnancy, although gestational thrombocytopenia may occur. Certain conditions can lead to coagulopathy such as intrauterine death, amniotic fluid embolism, sepsis, abruption and HELLP syndrome. Also, if a woman bleeds she may develop a clotting abnormality due to depletion and dilution of coagulation factors.

You are called to the obstetric ward urgently to a woman who has just had a vaginal delivery but is now bleeding significantly. What is your immediate management?

It is vital to work in a team with the midwives and obstetricians, and call for senior help promptly if required. Having established a brief history from the midwifery staff, the patient's notes and the obstetricians, I would need to establish how much blood had been lost, how much fluid had already been given and assess the current haemodynamic status. If the blood loss is more than 1,000 mL with ongoing bleeding and/or evidence of coagulopathy I would activate the major obstetric haemorrhage protocol, alerting haematology and portering services. I would give 100% oxygen, ensure the airway was patent and establish verbal communication with the patient. The blood pressure, pulse rate, oxygen saturation and capillary refill would need to be assessed and large bore intravenous cannulae inserted for further volume resuscitation. I would take blood for FBC, U&E, venous blood gas, coagulation studies, cross-match and point-of-care testing such as ROTEM or TEG if available. I would administer 1 g of tranexamic acid and initially resuscitate the woman with up to two litres of warmed intravenous crystalloid and consider a blood transfusion (depending on the extent of the haemorrhage, the clinical status of the patient and the haemoglobin concentration). I would be in constant

communication with the rest of the team to ascertain the extent of the bleeding and the planned treatment. This might include manual stimulation of an atonic uterus, placement of a urinary catheter and uterotonic drugs such as syntocinon, ergometrine and carboprost.

The obstetricians diagnose uterine atony and retained products of conception. What drugs do we use to aid uterine contraction?

The uterus contains a number of receptors that can be stimulated to cause contraction of its smooth muscle. These include oxytocin receptors, α_1 adrenoceptors, serotonergic and prostaglandin receptors. Syntocinon is a synthetic oxytocin analogue that acts to increase both the frequency and force of contractions. It does cause peripheral vasodilatation and so can induce hypotension and a reflex tachycardia. Ergometrine is an ergot alkaloid that acts via adreno- and serotonergic receptors to stimulate uterine contraction. Side effects include hypertension, coronary artery vasospasm and it is a potent emetic. Finally, prostaglandin analogues are used to stimulate uterine contraction. These include prostaglandin F_{2a}, or carboprost. The side effects of carboprost include bronchospasm, pyrexia, flushing and hypotension. It is contraindicated in patients with asthma.

All of these drugs are used in a stepwise manner when treating severe postpartum haemorrhage, in conjunction with surgical interventions.

What about other drugs that affect uterine contraction, tocolytics. What are they used for? Name me some examples and their mechanism of action.

Tocolytics are drugs that are used to inhibit uterine contraction. They may be indicated to inhibit premature labour or to reduce uterine contractions in cases of uterine inversion or cord prolapse. Drugs that are used can be classified according to their mode of action. Drugs may act as oxytocin receptor antagonists, calcium channel antagonists, beta-2 adrenoreceptor agonists or act via nitric oxide. Atosiban is a specific oxytocin receptor antagonist. Calcium channel antagonists such as nifedipine, magnesium sulphate and the volatile agents may be used. Drugs acting on β receptors include salbutamol, terbutaline and ritodrine (although this particular medication is no longer recommended because of its poor side effect profile). GTN may be used sublingually to relax uterine smooth muscle, acting as a nitric oxide donor.

The obstetric team are keen to move to theatre and surgically intervene. The woman continues to bleed. How would you proceed?

I would appreciate the urgency of the situation and attempt to move the woman quickly, while alerting the ODP and theatre team to prepare cell salvage and a rapid transfusion device. I would want to continue fluid resuscitation, consider invasive monitoring and have the woman mobilized in theatre. I would use point-of-care monitoring of haemoglobin, lactate and coagulation (TEG or ROTEM if available), provide rapid guidance on blood transfusion and advise whether products such as fibrinogen, FFP, or platelets should be administered. Cell salvage and attention to normothermia would also be considerations. In order to provide anaesthesia for removal of the products of

conception the options are a general or regional anaesthetic technique. My choice would depend on the urgency, haemodynamic stability of the patient and potential for coagulopathy.

What surgical interventions might be considered?

The obstetricians might only need to manually remove the placenta and stimulate the uterus for the bleeding to subside. If this fails, further options include uterine packing, uterine and internal iliac artery ligation, B-Lynch suture and finally a hysterectomy. In some units radiological embolization of pelvic vessels may be available.

You decide to proceed with a GA. What are your concerns with respect to the induction and maintenance of anaesthesia?

I would be primarily concerned about risks regarding the patient's airway, risk of aspiration, cardiovascular system and the impact of anaesthesia on uterine tone. I would call for senior help if I had serious concerns around any of these aspects.

Cases of failed intubation and difficult airway occur more frequently in obstetric cases. I would ask for a video laryngoscope to be prepared, consider use of a HELP pillow, ensure optimum position of the patient and ensure a pre-intubation checklist was run through, prior to an RSI intubation. The obstetric population desaturate more rapidly, so I would ensure good preoxygenation and consider apnoeic oxygenation with nasal cannulae or nasal high flow. Given the patient's blood loss, induction might affect cardiovascular stability. Therefore, alongside fluid resuscitation, I would have vasopressors ready, have invasive blood pressure monitoring if possible and use an opiate with my induction agent.

Excessive doses of volatile anaesthetic agents can contribute to uterine atony, so as well as being in constant communication with the obstetric team, I would monitor the dose being given to maintain anaesthesia, and potentially use nitrous oxide.

Further Reading

Drew T, Carvalho JCA. Major obstetric haemorrhage. *BJA Education*. 22 (6) 238–244, 2022.

Mavrides E, Allard S, Chandrachud E, Collins P, Green L, Hunt BJ, Riris S, Thomson AJ, on behalf of the Royal College of Obstetricians and Gynaecologists. Prevention and management of postpartum

haemorrhage. *BJOG: An International Journal of Obstetrics and Gynaecology*. 2016;124:e106–e149

MBRRACE-UK Saving lives, Improving mothers' care. *Lessons learned to inform maternity care from the UK and Ireland Confidential Enquiries into Maternal Deaths and Morbidity 2017–19*.

2.7.1 Orbital Anatomy – Sethina Watson

Describe the anatomy of the orbit

The orbits are pyramid-shaped bony cavities containing the globe, muscles, nerves, tissue and blood supply to the eyes. The base of the pyramid is directed towards the face with the apex directed posteriorly and medially. To protect the orbital contents the orbit has strong bony margins comprised of seven main bones (Figure 2.7.1.1).

Together these bones form the boundaries of the orbit:

- Roof – frontal bone (orbital plate) and lesser wing of sphenoid bone
- Floor – maxilla and orbital process of the palatine bone
- Medial wall – maxilla, ethmoid, lacrimal bone and body of sphenoid
- Lateral wall – zygoma, greater wing of sphenoid.

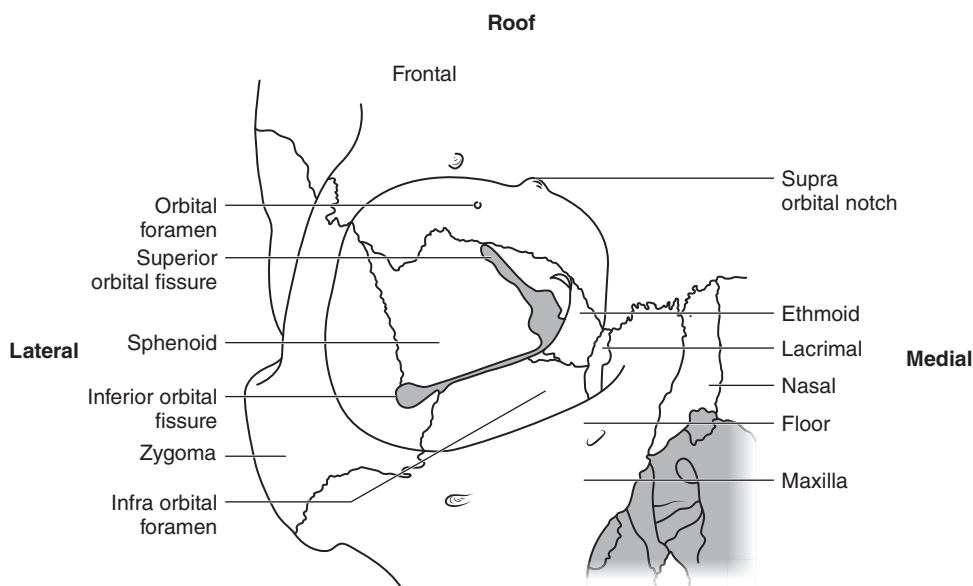


Figure 2.7.1.1 Bones of the orbit.

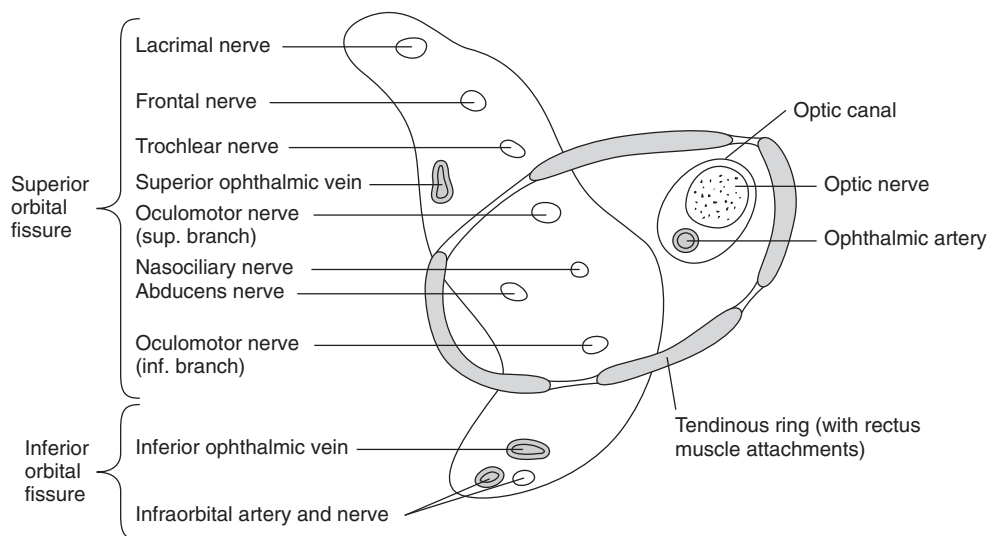


Figure 2.7.1.2 Structures passing through the orbital fissures.

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There are three main openings in the orbit:

- Superior orbital fissure (Figure 2.7.1.2)
- Inferior orbital fissure
- Optic canal (foramen).

The superior orbital fissure is a slit-like opening lying between the roof and the lateral wall. The fissure is divided into two parts by a fibrous ring (the annulus of Zinn) from which the main extraocular muscles originate. This origin can be described as a cone, the fibrotendinous ring making the apex and the four rectus muscles (superior, inferior, medial and lateral) forming the walls of the cone. Anteriorly, these muscles insert onto the sclera. There are two oblique extraocular muscles, superior and inferior, that originated from the sphenoid bone and maxilla. The levator palpebrae superioris muscle also arises from the tendinous ring and inserts into the upper eyelid. Several other important structures pass through the superior orbital fissure:

- Superior ophthalmic vein
- Oculomotor nerve (CN III)
- Abducens nerve (CN VI)
- Trochlear nerve (CN IV)
- Lacrimal, front and nasociliary nerves (branches of ophthalmic division of CN V).

The inferior orbital fissure transmits:

- Inferior ophthalmic vein
- Infraorbital artery
- Infraorbital nerve.

The optic canal is in the posterior roof and transmits the optic nerve CN II and ophthalmic artery.

Describe the nerve supply to the orbit.

Nerve supply to the orbit is motor, sensory and autonomic.

Motor supply to the ocular muscles is predominantly from the oculomotor nerve, superior oblique is supplied by the trochlear nerve and lateral rectus supplied by the abducens nerve.¹ The orbicularis oculi muscle is innervated by the facial nerve and levator palpebrae superioris via sympathetic innervation.

Sensory nerve supply is via branches of the trigeminal nerve:

- Frontal – supplies skin of upper eyelid, forehead and scalp
- Lacrimal – supplies lacrimal gland
- Nasociliary – sensory to cornea, conjunctiva and sclera (with additional supply from long and short ciliary nerves).

Autonomic nerve supply is important for pupil diameter, ocular blood flow and regulating intraocular pressure. The target organs are the sphincter pupillae and ciliary muscles:

- Sympathetic fibres – originate in first thoracic ganglion and synapse in the superior cervical ganglion. Causes iris dilation (mydriasis).
- Parasympathetic fibres – originate in Edinger-Westphal nucleus, preganglionic fibres travel with oculomotor nerve, then synapse in the ciliary ganglion and continue their postganglionic pathway via the short ciliary nerves. Causes iris constriction (miosis).

Describe the anatomy of the eyeball.

The globe is a roughly spherical structure occupying the anterior half of the bony orbit with an average axial length of 25 mm. The globe has three layers:

- Outer: tough fibrous layer, the opaque **sclera**. The sclera anteriorly becomes the transparent **cornea**.
- Middle: a vascular, pigmented layer containing the **choroid**, **ciliary bodies** and the **iris**.
- Inner: a delicate **retina**, consisting of an inner nervous layer and outer pigmented layer.

The globe itself can be divided into anterior and posterior compartments (Figure 2.7.1.3).

The anterior compartment contains everything anterior to the lens; it can also be divided again into anterior and posterior chambers.

The anterior chamber contains all the structures anterior to the iris. The iris itself contains the smooth muscle fibres for pupillary dilation and constriction. The posterior chamber contains the structures posterior to the iris but anterior to the lens (see Figure 2.7.1.3).

Both the anterior and posterior chambers in this anterior compartment contain aqueous humour. Aqueous humour originates in the posterior chamber of the eye and is secreted by the ciliary body and passes through a network of trabecular into the canal

¹ Hint for remembering this is the made up 'formula' $LR_6(SO_4)_3$.

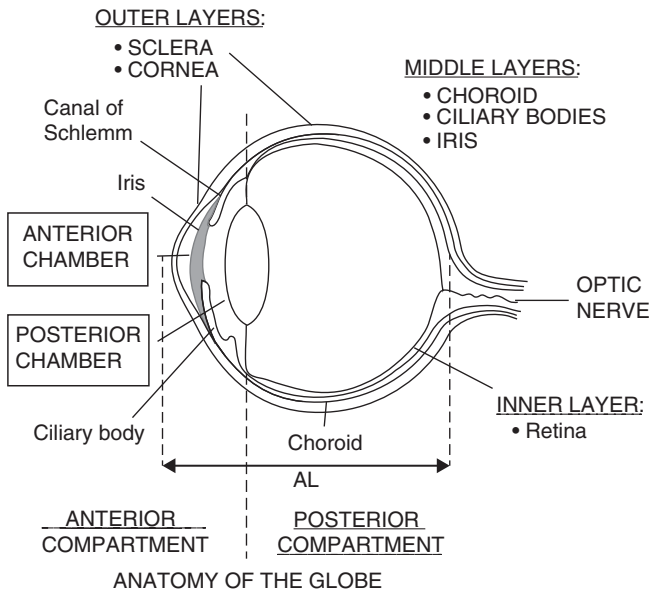


Figure 2.7.1.3 Anatomy of the globe.

of Schlemm where it is reabsorbed. Aqueous humour is a key component in maintenance of normal intraocular pressure. Blockage of this drainage can cause intraocular pressure to rise, known as glaucoma.

The posterior compartment behind the lens contains the gelatinous vitreous humour.

The retinal layer has an innermost layer of ganglion cells with axons that form the optic nerve and the macula, a small area responsible for central vision. Tenon's fascia surrounds the eyeball, except the cornea, it separates the globe from surrounding fat and orbital structures. Extraocular muscles and nerves penetrate this fascia at the corneoscleral junctions enabling eye movement. This is a potential space in which local anaesthetic can be infiltrated.

Describe the preoperative assessment of a patient presenting for ophthalmic regional anaesthesia.

Patients for ophthalmic surgery can present as an emergency or for elective surgery. Surgery spans all ages, from paediatric to the more common elderly population. Thorough preoperative assessment is required in all cases, many may have already been seen in preoperative assessment clinics. Many procedures are conducted as daycases, under local anaesthesia alone. Patients, especially the elderly, may have pre-existing comorbid conditions or uncommon medical problems resulting in ocular manifestations. Decisions for anaesthetic technique should be tailored to the individual and type of surgery.

Perioperative history.

I would conduct a full preoperative assessment regardless of whether they are having local anaesthesia, with or without sedation or a general anaesthetic. This would include

past medical history, previous anaesthetic history and previous surgeries, medications, allergies and social history. Medications such as anticoagulants and diabetic medications need careful planning for the perioperative period; I would consider involvement of the multidisciplinary team to discuss risk and benefits of treatment cessation. Elderly frail patients especially, will need assessment for mobility, planning for a safe discharge and assessment of risk of postoperative cognitive dysfunction. Paediatric patients also require thorough preoperative assessment involving parents or carers in order to examine any congenital or medical conditions.

Medical history should include assessment of cardiorespiratory disease that would limit the patient's ability to lie flat or neurological conditions causing movement disorders, tremor or confusion. Surgeries such as trabeculectomy, vitrectomy, limbal relaxation or complex lenses may be of longer duration. It is always prudent to consider general anaesthesia in patients where regional techniques may not be suitable.

Perioperative Examination.

I would check documentation of any preoperative examination or investigation done by the ophthalmologist, to assess suitability for regional technique. I would look for:

- Axial length. Lengths of >26 mm have an increased risk of globe perforation.
- Staphyloma, an outpouching of the globe. If >27 mm this presents an increased risk of globe perforation.

Perioperative Investigations and Examination

I would follow guidance for preoperative investigations. In particular, a clotting profile for those on anticoagulants, electrolytes and plasma glucose for those with renal or diabetic histories. On examination I would assess the eye looking for any issues that may make regional techniques more difficult, such as nystagmus or evidence of previous eye surgery.

What is the oculocardiac reflex?

This is a parasympathetically mediated bradycardia caused by pressure on the globe or traction on extraocular muscles. It is most seen in paediatric strabismus surgery. It warrants prompt attention and good communication with the surgeon. Rectifying measures include: releasing pressure on the globe, ceasing any stimulus, treating with anticholinergics, deepening of anaesthesia, or in severe circumstances, commencing CPR. The afferent pathway is via the trigeminal nerve to the medulla, with the efferent pathway travelling to the sinoatrial node via the vagus nerve.

Describe any preoperative guidance relating to anticoagulant or antiplatelet drugs.

Guidance has been agreed between the Royal College of Anaesthetists and Royal College of Ophthalmologists in 2012. Generally, the guidance states that these medications should be continued in the perioperative period for patients undergoing daycase cataract operations. Normal therapeutic targets should be maintained for the patient, as the risk of bleeding is outweighed by the increased risk of adverse thrombotic events such as stroke. International normalised ratio (INR) targets can be as high as 2.5 or even

3.5 in those with mechanical heart valves. For those having more complex surgery or where a haemorrhage adversely affects surgical outcome a multidisciplinary approach should be sought. I would also consult local guidance when indicated.

What are the contraindications to regional anaesthesia?

Contraindications can be classified into absolute and relative.

Absolute contraindications are patient refusal, allergy to local anaesthetic and localised sepsis of the globe.

Relative contraindications include:

- Inability to lie still in supine position
- Poor compliance with instructions
- Postural difficulties
- Confusion
- Communication difficulties
- Grossly abnormal coagulation
- Perforated globe or trauma.

What are the complications of regional anaesthesia?

Complications can be divided into common or rare.

Common complications include chemosis (swollen conjunctiva), subconjunctival haemorrhage and pain.

Rare complications include ecchymoses, retro-bulbar haemorrhage, globe injury, optic nerve atrophy, muscular palsy and brain stem anaesthesia. These can be sight or life-threatening. Sharper needles are associated with increased risk.

How would you perform a Sub-Tenon's block?

Consider developing your own general introduction that you can speak confidently and clearly.

I would take a thorough preoperative assessment, rule out any contraindications, discuss the procedure with the patient and obtain verbal consent. I would use a Prep-Stop-Block (or Stop-Before-You-Block) approach. The anaesthetic room would be prepared with the necessary equipment, emergency drugs, IV access gained, patient positioned, and site cleaned with routine monitoring applied. With a trained assistant we would confirm site, side marked and consent immediately prior to the block.

I would first topicalise the eye with local anaesthetic; blinking may encourage spread across the eye. The eye is then sterilised with drops of povidine-iodine solution.

Then I would apply a lid (Barraquer) speculum to keep the eye open. I would ask the patient to look up and out to expose the inferonasal quadrant of the eye. I would use non-toothed (Moorfields) forceps to take a small bite of the conjunctiva and Tenon's fascia about 5–10 mm from the limbus, avoiding any obvious blood vessels or pterygia. Using Westcott scissors I would make a small cut (around 2 mm) exposing the white scleral layer, the closed blunt tip of these scissors can be used to gently create a passage in this potential space. A Sub-Tenon's needle with syringe of local anaesthetic attached is gently introduced along the contour of the eyeball. The syringe should be vertical and at a depth of 15–20 mm before I inject 3–5 ml of local anaesthetic solution posterior to the

globe; 2% lidocaine or 0.5% levobupivacaine has been used, sometimes with hyaluronidase as an additive. I would aspirate prior to injection to avoid intravenous spread. I would remove the needle, close the eye and gently massage the eye to enable spread of the local anaesthetic. I would then assess for adequacy of the block with muscle akinesis and monitor for complications.

Describe the peribulbar technique.

Use similar introduction to above method. Use of peribulbar techniques may be less common now that Sub-Tenon's blocks are more widespread. If you have not performed this block, you may describe how you have seen it performed.

Following topicalisation of the eye with local anaesthetic and sterilisation using povidine-iodine solution, I would ask the patient to look straight ahead. In the far inferotemporal corner of the eye a perconjunctival injection is made. The needle is directed parallel to the orbital floor, and passed posteriorly. The needle tip should be kept extraconal. I would then aspirate before injecting around 5–10 ml of local anaesthetic. The patient may report a pressure headache, which should resolve after several minutes. I would assess block adequacy via akinesis and consider whether any supplemental block may be needed for the surgery. I would remain vigilant for any complications.

Further Reading

Anker R. Regional anaesthesia for ophthalmic surgery. *BJA Education*. 17 (7): 221–227 (2017).

Kong KL, Allman K, Budd J. Royal College of Anaesthetists. Chapter 13. Guidelines for the Provision of Anaesthesia Services (GPAS). Guidelines for the Provision of Ophthalmic Anaesthesia Services 2020.

2.7.2 Control of Intraocular Pressure – Sethina Watson

What is a normal intraocular pressure?

Intraocular pressure (IOP) is the tissue pressure of the intraocular contents on the sclera. The normal range is between 10 mmHg and 20 mmHg. Generally, it increases with age but is maintained within this range for much of adult life. There is some diurnal and postural variation. Pressure can increase transiently in situations such as coughing or straining.

What factors determine IOP?

The globe is essentially a non-compliant spherical structure within a rigid bony cavity (a similar concept to the brain inside the skull). Factors determining IOP can be intraocular or extraocular.

Intraocular factors are predominantly related to a change in volume of aqueous humour (production and drainage), blood flow (arterial and venous) and other causes of raised IOP within the globe such as a mass or foreign body. Aqueous humour regulation is the main determinant of IOP. Vitreous humour is normally of fixed volume and has minimal effect on IOP. As a person ages the sclera becomes less compliant and IOP can increase.

Extraocular factors relate to compression from retrobulbar haematomas, tumours or insertion of local anaesthetic in regional blocks and increased extraocular muscle tone. In the perioperative period external pressure can be caused by patient position (especially prone), face masks and direct pressure from surgeons.

How is intraocular pressure regulated?

IOP is normally regulated by aqueous humour production and drainage (Figure 2.7.2). Aqueous humour is a clear fluid that fills the anterior and posterior chambers of the eye. There is also a minor contribution to fluid via ultrafiltration of plasma. Aqueous humour is produced at a constant rate in the ciliary bodies via active secretion. It flows through the iris into the anterior chamber from the posterior chamber. It is then reabsorbed via the canal of Schlemm in the angle between the iris and cornea. In normal circumstances inflow (production) should equal outflow (drainage) and IOP is maintained. Usually if intraocular pressure increases there is a corresponding increase in reabsorption. However, if reabsorption is blocked (for example, increased resistance caused by glaucoma) or episcleral venous pressure is increased (for example, by increased central venous pressure (CVP)) then outflow is compromised and IOP rises.

Why is IOP of relevance to anaesthetists?

Patients can present in the emergency and elective setting. Penetrating eye injuries can increase IOP causing expulsion of globe contents with loss of vision. For elective ophthalmic procedures, patients may present with acute or chronically raised IOP. For non-ophthalmic surgery patients may present with chronically raised IOP. In the perioperative period many of the drugs we use affect IOP and also effects caused by regional techniques, positioning, as well as stimulating physical interventions such as laryngoscopy and intubation. It is important to understand our role in minimising risk of ocular damage or sight loss.

What is the role of blood volume and IOP?

Venous pressure most strongly affects IOP; normal venous pressure within the globe is just above IOP at around 15 mmHg. Raised central venous pressures from coughing, straining, Valsalva manoeuvres or vomiting cause venous congestion which can raise intraocular volume. This increase in volume then impairs episcleral venous drainage causing increased IOP.

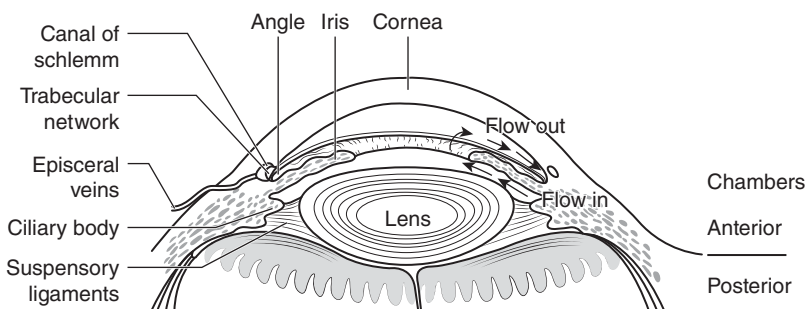


Figure 2.7.2 Drainage of aqueous humour.

Arterial blood supply has some autoregulation via retinal and choroidal circulations and is affected by low systemic pressure reducing IOP. Like cerebral blood flow, arterial flow is related to partial pressures of carbon dioxide and oxygen. Hypercarbia, hypoxaemia and increased metabolic rate cause increased blood flow and increased IOP.

How can anaesthetists avoid unwanted rises in IOP?

The most important influence an anaesthetist can control is avoidance of raised episcleral venous and CVP. This can be achieved by physical and pharmacological means.

In the perioperative setting physical methods such as head-up tilt, neutral head position, avoidance of neck ties and minimising coughing and straining can help reduce IOP. Coughing, straining and vomiting can increase IOP by 30–40 mm Hg. Laryngoscopy and intubation can increase IOP by 10–20 mmHg. Consideration of supraglottic devices in appropriate patients and use of pharmacological methods to reduce response to laryngoscopy.

Pharmacological methods specific to decreasing IOP include topical medications to optimise aqueous humour drainage (parasympathomimetics such as cholinergics and anticholinesterases) and reduce aqueous humour production (sympathomimetics such as beta antagonists including timolol). Use of systemic medications such as acetazolamide (reduces aqueous humour production) and mannitol (osmotic diuretic acting on vitreous humour) can reduce IOP.

How do anaesthetic drugs affect IOP?

Drugs that we commonly use for induction or muscle relaxation affect IOP. They can be divided into drugs that increase or decrease intraocular pressure.

All induction agents except for ketamine reduce intraocular pressure. All inhalational agents reduce IOP.

Non-depolarising muscular blockers (NDMB) decrease extraocular muscle tone and thus decrease IOP. Suxamethonium transiently increases IOP; however, it is usually given after an IOP reducing induction agent. It also reduces coughing and facilitates rapid intubating conditions. Assessing airway versus eye risk is key and suxamethonium use may be appropriate in emergency situations, although rocuronium in higher doses is increasingly used.

Other relevant drugs include opioids and antiemetics. Opioids do not directly affect IOP but are useful in obtunding the hypertensive response to laryngoscopy. Antiemetics should be used prudently to avoid postoperative nausea and vomiting.

Nitrous oxide should be avoided. It has no effect on IOP but can expand gas-filled spaces. In some ophthalmic surgery, such as retinal detachment repair, sulphur hexafluoride is used. Nitrous oxide causes complications of this gas bubble and may lead to retinal detachment.

Describe your ideal anaesthetic for surgery in patients with an open eye injury

In these patients a general anaesthetic enables better control of factors affecting IOP, such as carbon dioxide and oxygen partial pressures. I would aim for the following:

- Smooth induction with muscle relaxation

- Careful placement of endotracheal tube or supraglottic airway, avoiding hypertensive responses and coughing
- Ventilation, enabling me to control PaO₂ and PaCO₂
- Careful attention to positioning, consideration of a head-up tilt and avoidance of neck ties
- Smooth extubation
- Judicious prescribing of antiemetics to reduce postoperative nausea and vomiting.

The surgeon states that the globe contents are protruding. What can be done to reduce IOP acutely?

This is an emergency that needs prompt attention.

I would assess the patient systematically starting with a rapid assessment of airway and breathing, ensuring that they are well oxygenated and not hypercapnic. With regards to circulation, I would ensure that their blood pressure is within normal range and check that nothing impairs venous drainage. There should be no tube ties, the head in neutral position and a head-up tilt could be used. I would check that the patient is not coughing or straining. I may need to increase depth of anaesthesia or use a non-depolarising neuromuscular blocker. Intravenous propofol is effective in reducing intraocular pressure quickly.

Drugs such as mannitol and acetazolamide can be used. If these drugs are used, the patient should be monitored postoperatively for electrolyte abnormalities (seen with acetazolamide use) or ongoing side effects of mannitol-induced diuresis.

Further Reading

Murgatroyd H, Bembridge J. Intraocular pressure. *Continuing Education in*

Anaesthesia Critical Care and Pain. 8 (3): 100–103 (2008).

2.7.3 Penetrating Eye Injury – Jade A Loughran and Sarah F Bell

This is a topic that has come up many times. It is a question that can be used to test a number of different areas including: your physiological and pharmacological knowledge; your ability to weigh up the pros and cons of immediate surgical intervention versus the risk of aspiration; and your appreciation of the importance of communication with the rest of the surgical team. It is vital to indicate to the examiner that you have a solid knowledge base and appreciation of all of these problems.

You are the anaesthetic registrar on call and have been called to the accident & emergency department to see a 20-year-old man. He has been brought in by ambulance having sustained significant facial injuries after being involved in a fight outside a pub.

What would be your initial actions?

Remember to try and picture yourself in this situation. There may be more going on than just the facial trauma. It may be worth making an initial statement to show the examiner that you appreciate this.

I would assess this patient as a trauma patient, taking an ABCDE approach and initiating treatment as any problems are found. If there were other members of the

emergency department available I would ask them to assist me in obtaining baseline observations and intravenous access. Given the history I would be considering the possibility of airway and breathing complications, circulatory compromise due to haemorrhage, neurological change due to possible head injury, alcohol or drugs, and hypothermia. It is important to remember that there may be more injuries than just the obvious facial trauma.

My assessment of the airway would involve trying to talk to the patient and then listening for sounds of airway obstruction or compromise. If there were any concerns of airway compromise I would consider immediate further interventions as required including suction, airway adjuncts and intubation. I would apply high-flow facial oxygen to the patient. Next, I would examine the respiratory system looking at the respiratory rate, oxygen saturations, observing and auscultating the chest. My circulation assessment would include measurement of the heart rate and blood pressure. I would attempt to identify any major sources of bleeding (which might be facial in this patient) and reduce any haemorrhage. I would examine the abdomen and long bones. My neurological assessment would include the Glasgow Coma Score and a brief general assessment of peripheral neurology. If possible, I would assess the response of the pupils to light. I would then check the glucose and temperature. After this initial primary assessment, it would be important to consider what investigations or interventions are required at this stage, and to consider a secondary survey when appropriate.

You find that there are numerous bleeding facial lacerations. The patients' left eye has been damaged by a glass bottle. You cannot find any other injuries and the patient is fully conscious and cooperative. What would be your next steps?

My priorities would still be the airway, breathing and circulation. From your description the airway and breathing are stable at present and so I would concentrate on the circulation and attempt to halt the bleeding. I would ask one of the accident and emergency staff to exert pressure on the wounds and possibly suture the lacerations or bleeding points. I would obtain wide-bore intravenous access and send off baseline bloods. Analgesia, tetanus and antibiotics may be required. I would contact the ophthalmologist on call to come and urgently review the eye injury. Depending on the extent of the facial lacerations, I may also need to contact a plastic, general or maxillofacial surgeon.

The ophthalmology registrar attends promptly and is extremely keen to take the patient to theatre urgently because he has a penetrating eye injury. What further information would you want to know from the patient before proceeding to theatre?

I would need to ensure that the patient was haemodynamically stable and that their airway and breathing did not require assistance. I would also want to take a full history from the patient. Specifically, I would want to know the details surrounding the incident, whether a head injury could still be a possibility, whether the patient had consumed any

alcohol or other substances and whether they had sustained any other injuries. I would also want to take a general history of the patient's past medical conditions, anaesthetic history, drug history, allergies, starvation history and dentition. I would need to formally assess the airway for intubation.

Finally, I would discuss the case with the surgeon and the consultant anaesthetist on call. I would want to know the planned operative procedure, the indication for emergency surgical intervention and whether this outweighed the aspiration risk posed by the patient with a potentially full stomach.

The ophthalmologist wants to take the patient to theatre to remove a piece of glass that is lodged in the patient's left eye. The maxillofacial surgeon is happy to then proceed to suture the facial lacerations and is confident that there are no facial fractures present. Is there anything else you might consider before theatre?

I would discuss whether a CT head scan might be required to assess whether the glass had migrated intracranially and to rule out any concurrent head injury, especially if the history suggested this was a possibility.

Furthermore, I would want to premedicate the patient with a prokinetic (such as 10 mg metoclopramide given intravenously), a drug to reduce the gastric acidity (such as 50 mg of ranitidine IV or omeprazole) and an acid-neutralising medication (such as 30 ml of 0.3 M sodium citrate taken orally).

Let's think about some of the physiological considerations in this case. Can you tell me the normal range for intraocular pressure?

Yes, this is 10 to 20 mmHg.

What determines intraocular pressure?

The globe is a non-compliant sphere within a rigid bony box. The intraocular pressure will therefore change if pressure is exerted on the eyeball itself or if the volume of the contents of the orbit changes.

How is intraocular pressure normally regulated?

The main regulation is from the volume of aqueous humour in the anterior chamber; a rise in intraocular pressure will be compensated for to some degree by an increased rate of aqueous humour drainage, but adaptation takes 15–30 minutes to occur. The vitreous humour in the posterior chamber has a relatively fixed volume and so plays a minimal role in regulation of intraocular pressure.

How is aqueous humour produced and where does it get reabsorbed?

Aqueous humour is produced by the ciliary body to supply glucose and oxygen to the lens and cornea. It flows from the posterior chamber to the lens and iris and is then reabsorbed in the anterior chamber, through the trabecular network and the canal of Schlemm (in the angle between the iris and the cornea). The fluid then flows into the

episcleral veins. Drainage is dependent on the pressure gradient between the chamber and the veins.

How is intraocular pressure increased?

Intraocular pressure may be affected by aqueous humour drainage, the arterial blood supply to the eye and certain drugs.

Intraocular pressure is increased if drainage of aqueous humour is reduced, either by a reduction in the drainage system (for example glaucoma) or because of an increase in venous pressure.

Choroidal arteries differ from retinal arteries in that they do not have myogenic autoregulation. They will dilate in response to increased perfusion pressure, hypercarbia or hypoxia. Straining, vomiting and coughing will increase blood pressure and thus perfusion pressure. All of these factors will therefore cause an increase in intraocular pressure.

Drugs such as ketamine and suxamethonium are known to transiently increase the intraocular pressure, although this is only for 15 to 20 minutes duration.

Why is it important to avoid increases in intraocular pressure in this patient?

An increase in intraocular pressure may lead to expulsion of the globe contents via the traumatic opening. This will cause further injury to the eye. It is therefore important to understand the mechanisms for increased ocular pressure so that they can be avoided.

Let's now return to our case history. You have agreed to anaesthetise this man for an emergency ophthalmic operation. His facial lacerations have been temporarily dressed and have stopped bleeding. He is in the anaesthetic room and has had premedication as discussed. Can you describe to me how you would induce anaesthesia and the potential pitfalls that might be encountered?

The ideal induction would avoid hypoxia, hypercarbia, hypertension, coughing, vomiting and straining as these will all increase intraocular pressure. The risk of aspiration needs to be considered as does the need for endotracheal intubation, since laryngoscopy may also increase intraocular pressure.

This patient potentially has a full stomach. Their gastric emptying time may also be delayed by the effects of pain and opioid analgesia. I would therefore use a rapid sequence induction technique and secure the airway with an endotracheal tube. Prior to inducing anaesthesia I would ensure that I had resuscitation equipment and drugs available, a trained assistant, I would check the anaesthetic machine as per the AAGBI guidelines and I would have a tilting table. I would preoxygenate the patient for 3 minutes with a tight-fitting face mask. I would have suction and difficult airway equipment available. Once my assistant had placed cricoid pressure I would induce anaesthesia with thiopentone 5 mg/kg and suxamethonium 1–2 mg/kg. I would expect the reduction in cardiac output due to the induction agent to offset the increase in intraocular pressure

caused by the suxamethonium. I would therefore aim to maintain a normal intraocular pressure during induction. I would not use ketamine because it would increase intraocular pressure.

Could you use a total intravenous technique for induction and maintenance of anaesthesia?

Yes, you could use an intravenous propofol infusion to induce or maintain anaesthesia. This might be advantageous due its antiemetic properties. I would still use cricoid pressure and suxamethonium as part of a modified rapid sequence induction with this technique.

What drugs might you consider to reduce the increase in intraocular pressure during induction?

The increase in intraocular pressure might be reduced by a beta-blocker or short-acting opioid such as alfentanil. Rapid reduction of intraocular pressure might be achieved by acetazolamide which reduces aqueous humour production, or mannitol which acts as an osmotic diuretic.

How would you manage the patient intraoperatively?

I would aim to maintain normal oxygen and carbon dioxide levels. I would keep the blood pressure and temperature within normal limits. I would administer a combination of antiemetics such as ondansetron and dexamethasone in order to try and reduce the risk of nausea and vomiting postoperatively. I would also administer analgesics titrated to the expected pain caused by the operation. I would position the patient supine or slightly head up and avoid tight neck ties to optimise venous drainage.

How would you extubate the patient?

Extubation poses further risk of raising intraocular pressure due to coughing, straining or vomiting. This may not be a problem if the eye wound has been repaired, unless the insult has the potential to cause elevated pressures postoperatively. The effect of an elevation in intraocular pressure needs to be balanced against the potential risk of aspiration.

I would aim to achieve a smooth emergence with no coughing, straining or vomiting. This might be performed by extubating the patient deep or exchanging the endotracheal tube for a laryngeal mask airway (also while deep). Alternatively, beta-blockers or lidocaine could be given intravenously to reduce the hypertensive response. Personally, I would extubate the patient wide awake in a semi-recumbent position if I considered the aspiration risk to be significant.

Would a local anaesthetic technique be possible for this operation?

Penetrating eye injuries are almost never suitable to be done under local anaesthesia. This is due to the nature and duration of the surgery. Furthermore, local anaesthetic techniques can cause additional rises in intraocular pressure and the actual spread of the solution might be unreliable. A potential advantage of a local anaesthetic technique is that it would considerably reduce the risk of aspiration.

Further Reading

Murgatroyd H. Intraocular pressure.

Continuing Education in Anaesthesia

Critical Care and Pain. 2008;8(3):100–103.

2.7.4 Cataract and Detached Retina – Alice Braga

What factors need to be considered when selecting the appropriate anaesthetic for a patient presenting for cataract surgery?

Cataract surgery is the most commonly performed surgery in the NHS. In principle it can be performed under topical anaesthesia, Sub-Tenon's, peribulbar, retrobulbar, and general anaesthesia. The choice of anaesthetic depends on patient factors and ophthalmic factors, as well as surgical preference and locally agreed patient pathways including the availability of an anaesthetist. Around half of all cataract procedures are performed in the independent sector, without the presence of an anaesthetist, under topical anaesthesia. However, topical anaesthesia does not block sensation arising from intraocular structures in the anterior segment. Additional anaesthetic instilled into the anterior chamber, so called intracameral anaesthesia, improves patient comfort. Local anaesthetic blocks offer effective analgesia and have the advantage of providing akinesia of the eye. Factors that increase the likelihood of intraoperative pain include young age, surgery on the dominant eye, previous surgery, myopia, prolonged or difficult surgery, and accidental stimulation of the iris.

Only around 4% (4.4% National Ophthalmology Dataset 2020) of cataract operations are performed under general anaesthetic. Typically, general anaesthesia is reserved for adults who are unable to communicate, cooperate, or remain stationary during eye surgery, and for children. As with all patients undergoing general anaesthesia these patients will require appropriate preoperative preparation.

To what degree do patients require pre-optimisation for cataract surgery?

Patients presenting for cataract surgery tend to be elderly and often have complex comorbidities. In general, however, stable patients with ASA physical status classification I–III are suitable for surgery under local anaesthetic provided they can tolerate positioning to perform the procedure. Performance of a focussed medical history, including use of any anticoagulant or antithrombotic therapies, previous eye operations, and overall suitability for same-day surgery should be performed prior to day of surgery to minimise the risk of last-minute cancellations. Cataract surgery incurs little risk since physiological stress is minimal and no blood loss or fluid shifts occur. In contrast, significant delays for cataract surgery are associated with increased morbidity due to greater likelihood of falls with hip fracture, car accidents, worsening cognitive impairment, and higher mortality. Cataract surgery should not be postponed unless the patient has an acute condition that requires time to achieve optimal medical management. Examples might include myocardial infarction within 30 days, recent percutaneous coronary intervention (within 14 days without stenting or within 30 days with stenting), new onset of a clinically

significant arrhythmia, decompensated heart failure, acute respiratory conditions, recent stroke, malignant hypertension or recent episode of diabetic ketoacidosis. Patients should be advised to continue all medications for chronic conditions including anticoagulants. Patients receiving warfarin should continue. Surgery can be safely performed with the patient's INR in the treatment range for their condition.

A patient presents for cataract surgery under local anaesthesia. The admitting nurse informs you the blood pressure reads 176/105 mmHg. How would you proceed?

Pre-existing hypertension is the most common chronic condition in patients presenting for cataract surgery, with approximately half of patients receiving treatment for the disease. On-the-day measurements of blood pressure are often a reflection of patient anxiety and not a true measure of blood pressure risk. Patients undergoing cataract surgery are often on high-flow surgical pathways arriving only shortly before their appointment time with minimal time to recover from their journey to hospital. I would refer to the blood pressure reading taken at pre-assessment or GP records. Provided the patient feels well, if the blood pressure is elevated but not above 180/110 mmHg they should be advised to see their GP but surgery can continue. The patient and staff can be reassured that there is no additional risk of cardiovascular or neurological events. There is also little association between hypertension and surgical difficulty or haemorrhagic complications.

A 36-year-old Type 1 diabetic patient presents for surgery for a retinal detachment. The staff tell you she is a regular attender, and this is not her first procedure on this eye. What are your immediate thoughts regarding management of this case.

The considerations in this case include patient factors and surgical factors. The patient is young and has had previous eye surgery indicating there is an increased risk of intra-operative pain due to young age and possible anxiety. Microvascular disease of the retina suggests diabetic control is poor so I would want to ensure stability of management by checking the most recent HbA1C for the patient, ensuring they are feeling well, and that all routine diabetic medicine has been taken.

Patients undergoing vitreoretinal surgery usually receive a regional anaesthetic block and/or general anaesthesia rather than topical anaesthesia. Topical anaesthesia alone is avoided because surgery may be quite lengthy, and it is imperative that the patient does not move.

The presence of small vessel disease in the eye is a predictor for cardiovascular disease discouraging the use of general anaesthesia and its associated cardiovascular risk. Rotation of the globe with traction on the extraocular muscles during retinal detachment operations may elicit the oculocardiac reflex. Thus, vigilance must be maintained to detect bradycardia and other arrhythmias.

Patients who have undergone previous retinal detachment surgery can present a challenge to the anaesthetist. Scleral bands make Sub-Tenon's anaesthesia difficult or impossible and previous Sub-Tenon's anaesthesia can cause scarring and adherence of

the conjunctiva and tenons capsule to the sclera making access and anaesthetic spread challenging. Diabetic patients have thickened conjunctiva (squamous metaplasia) which alters the 'feel' of performing a Sub-Tenon's block. Good communication with the surgeon as to the exact nature of previous surgical interventions and presence and location of any bands is essential. A superomedial or even superolateral approach to Sub-Tenon's anaesthesia is possible. Peribulbar or retrobulbar techniques are the alternative but need to be performed by clinicians familiar with the technique due to the increased risk of complications with sharp needle techniques. Vitreoretinal surgery is often time-critical particularly in the case of 'macular on' detachment or trauma. The requirement to optimise patients is often offset by the urgency of the procedure. In the case of diabetes a pragmatic approach needs to be adopted to blood sugar management. If blood sugar is less than 4 mmol/L then patients should be offered something sweet with the aim to proceed with surgery. A high BM on the day of surgery should not result in cancellation of surgery unless there are concerns about hyperosmolar hyperglycaemic state (levels over 33 mmol/L) or ketoacidosis (BM greater than 13.9 mmol/L with evidence of ketones in blood or urine).

You are supervising a CT3 on call. A 56-year-old man presents for open reduction and internal fixation of his left ankle on the trauma list. He tells you that he recently underwent surgery for a retinal detachment. You discuss the anaesthetic plan with your trainee who wishes to use nitrous oxide for its analgesic properties and second gas effect. What are your concerns?

Intraocular air or gas is often used as a tamponade agent at the end of vitrectomy surgery to secure the retina. Intraocular gas only remains in the eye for a short period and is then resorbed but, while the gas is present in the eye, there are certain precautions that need to be taken to avoid harm. Anaesthetists need to be aware of the risks associated with the use of nitrous oxide. Nitrous oxide has a high diffusion and solubility coefficient causes it to diffuse down its concentration gradient more rapidly than nitrogen, oxygen, or carbon dioxide. Nitrous oxide diffuses into closed spaces with resultant increase in pressure. In an eye with intraocular gas the net effect is an expansion of the gas bubble that leads to a rapid increase in intraocular pressure with subsequent risk of reduced perfusion of the retina causing central retinal artery occlusion resulting in ischaemia and infarction. Nitrous oxide is used in several healthcare settings (out of hospital, A&E, theatres, and delivery suite) as well as being a drug of abuse. Patients should be clearly instructed about the risks of nitrous oxide and patients' medical records should carry a clear warning of the need to avoid nitrous oxide as part of the anaesthetic.

Further Reading

Hindy NEL, Johnston RL, Jaycock P, Eke T, Braga AJ, Tole DM, Galloway P, Sparrow JM, UK EPR User group. The Cataract National Dataset Electronic Multicentre Audit of 55567 operations: Anaesthetic

techniques and complications. *Eye*. (2009) 23, 50–55.

Kumar CM, Seet E, Eke T, Joshi GP. Hypertension and cataract surgery under loco-regional anaesthesia: Not to be ignored? *British Journal of*

Anaesthesia. 1027; 119 (5): 855–859.

Kumar CM, Seet E, Eke T, Joshi GP. Perioperative considerations for sedation-analgesia during cataract surgery: A narrative review. *Anaesthesia*. 2019; 74: 1601–1610.

The Royal College of Ophthalmologists Ophthalmic Safety Alert – Do not use nitrous oxide when there is gas in an operated eye.

The Royal College of Ophthalmologists Ophthalmic services document. High Flow Cataract Surgery. Version 2.0: VS January 2022.

2.7.5 Strabismus – Joy M Sanders and Zoe Riddell

You have been asked to anaesthetise a 4-year-old child for correction of a squint.

What factors should be considered in the preoperative assessment?

In addition to the general assessment of the child, it is important to consider whether this is an isolated strabismus or whether it is associated with a rare congenital syndrome such as Cruzon's or Apert's as these can be associated with learning difficulties, cardiac or craniofacial anomalies. Malignant hyperthermia was thought to also be associated with strabismus; however, this is unproven.

General considerations for paediatric anaesthesia include a history of medical conditions such as asthma, recent respiratory tract infections, epilepsy, or a history of prematurity. Other factors to consider include previous anaesthetic history, family history of anaesthetic problems, medications, allergies, and period of starvation.

The child and parents must be prepared for the conduct of anaesthesia, involving the child where appropriate. The presence of a parent in the anaesthetic room and afterwards in recovery should be discussed. Consent should be taken for suppositories and a topical local anaesthetic for cannulation, such as Ametop, should be applied. Pharmacological premedication, such as oral midazolam, should be considered if difficulties with induction are anticipated. The parents should be warned of the risk of postoperative vomiting, which is high in strabismus surgery, but it should be explained that measures will be taken to reduce this risk, although it cannot be eliminated completely.

So how would you anaesthetise this child?

I would ensure I knew the child's weight and calculate all drug doses accordingly. Full monitoring according to the AAGBI standards is essential. I would have 20 mcg/kg of atropine drawn up and immediately accessible due to the high incidence of the oculocardiac reflex. A prophylactic dose of glycopyrrolate is now not recommended.

Induction can be IV or inhalational depending on the preoperative discussion with the parents and child, and their cooperation in the anaesthetic room.

An armoured LMA can be used, or the child can be intubated using either a south-facing RAE, in children over one year, or a reinforced tube in infants. Factors favouring intubation would include a child under the age of one or a potential difficult airway. If an LMA is used, it is essential it is correctly seated, as access to the airway during surgery is limited. It is acceptable to use controlled or spontaneous ventilation; however, my preference is to use controlled ventilation, to avoid hypercarbia, which can increase the incidence of the oculocardiac reflex.

Additionally, many strabismus surgeons request complete muscle relaxation to enable them to perform the 'forced duction test' whereby the eye is moved to assess

any mechanical restriction in movement as distinct from that attributable to muscle tone. If the surgeon is placing an 'adjustable suture' the muscle relaxant needs to have completely worn off prior to adjustment of the suture. My relaxant of choice would be atracurium as it is not only short-acting, but the oculocardiac reflex is thought to occur less frequently with this agent compared to rocuronium.

I would use a mixture of oxygen and air with a volatile agent for maintenance of anaesthesia. I would avoid nitrous oxide if possible as it is known to increase the already high incidence of postoperative nausea and vomiting (PONV) associated with this surgery. However, I could also consider total intravenous anaesthesia to further reduce the risk of PONV especially if the child is over the age of 6, where vomiting starts to become more troublesome.

You mentioned the oculocardiac reflex. Can you tell me more about this reflex, the afferent and efferent pathways?

This is when surgical traction on the extraocular muscles (particularly the medial rectus) or pressure on the globe induces a reflex bradycardia, asystole or junctional arrhythmia. It can occur in up to 60% of strabismus surgery cases. It reverts almost immediately when the stimulus is removed.

Afferent pathways are from the long and short ciliary nerves, via the ciliary ganglion, to the ophthalmic division of the trigeminal nerve. They are transmitted to the trigeminal (Gasserian) ganglion and finally to the main sensory nucleus of the trigeminal nerve, in the floor of the fourth ventricle.

The efferent pathway is via the vagus nerve. It starts in the nucleus of the vagus in the cardioinhibitory centre of the medulla and runs to the heart. Increased stimulation leads to decreased cardiac output (See Figure 2.7.5).

Risk factors include hypercarbia, hypoxia, light plane of anaesthesia and use of opioids.

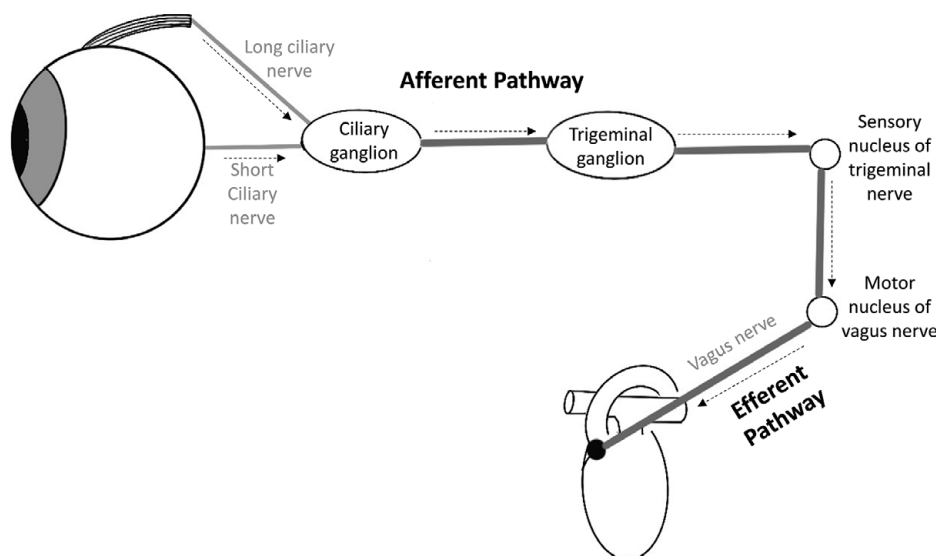


Figure 2.7.5 Oculocardiac reflex.

OK, so let's imagine that halfway through the operation you notice the heart rate has dropped to 50 beats/min. What would you do?

Having quickly confirmed the rate, I would promptly ask the surgeons to remove the stimulus. This is usually all that is required, because the reflex fatigues and is not as powerful the next time. However, if the heart rate does not improve, I would call for senior help, and administer 20 mcg/kg of atropine. This can be repeated if it has little effect. If cardiac output is lost, I would start cardiopulmonary resuscitation.

What would you use for intraoperative and postoperative analgesia for squint surgery?

This type of surgery is not particularly painful, and long-acting opioids should be avoided to decrease the risk of postoperative nausea and vomiting. A combination of fentanyl with a diclofenac suppository 1 mg/kg and intravenous paracetamol 15 mg/kg is usually adequate. Supplemental local anaesthesia should be used, and subconjunctival local anaesthetic has a longer-lasting effect than eye drops. An ophthalmic block can also reduce the risk of the oculocardiac reflex if performed before the strabismus surgery. Peribulbar blocks in theory could be used, but the risk of globe perforation and retrobulbar haemorrhage in children is higher than in adults, making this inadvisable.

You mentioned postoperative nausea and vomiting. Is this usually a problem?

Yes, the incidence of this can be greater than 50%. It is worse in bilateral operations. The precise mechanism is not known but may be due to the oculo-emetic reflex involving the ophthalmic division of the trigeminal nerve and the vomiting centre in the medulla. If the child experiences the oculocardiac reflex, they are likely to experience significant postoperative nausea and vomiting too.

It can be prevented through general measures such as ensuring adequate hydration with intravenous fluids. Two antiemetics should be given such as 0.15 mg/kg of ondansetron in combination with 0.15 mg/kg dexamethasone. As mentioned previously, avoidance of the oculocardiac reflex and total intravenous anaesthesia can further reduce the risk.

Further Reading

Chua AWYN, Chua M, Leung H, Kam PCA.
Anaesthetic considerations for strabismus

surgery in children and adults. *Anaesthesia and Intensive Care*. 2020; 48(4): 277–288.

2.7.6 Ophthalmology Local Anaesthetic Techniques – Charlotte Morris

What are the preoperative considerations for ophthalmic regional anaesthesia techniques?

A large proportion of ophthalmic surgery is for ophthalmic manifestations of systemic disease; therefore many patients presenting for surgery will have pre-existing medical

conditions. Patients should be seen in preoperative clinic for assessment and optimisation prior to surgery. The pre-op assessments should include comorbidities, medications, allergies, previous anaesthetic history and social history. Relevant investigations such as a 12-lead ECG should be carried out to assess those patients who have cardiorespiratory disease. Fasting may not be required if the patient is undergoing orbital regional anaesthesia, which may be of benefit to patients with diabetes. Many of these surgeries are performed as daycase procedures, and most cases are not required to be performed urgently, so patients should be optimised as much as possible.

Ophthalmic history and orbital examination is performed to assess suitability for local anaesthetic technique. The orbit should be visually inspected for signs that may suggest difficult regional approach such as enophthalmos, nystagmus, narrow palpebral fissure and previous surgery to the area. The axial length of the globe needs to be checked (normal length around 23 mm). If the axial length is >26 mm or the patient is a long-standing myopic with where the eye becomes more elongated they may have thinning of the sclera and staphylomata (outpouchings of the globe). This increases the risk of perforation of the globe.

Discussion with the patient about the anaesthetic technique and the positioning required allows the anaesthetist to assess suitability for regional technique as well as gain consent from the patient. Patients who are undergoing trabeculectomy, vitrectomy, limbal relaxation or difficult lenses are likely to have a longer surgery; this will have implications on the type of regional block used and also the ability of the patient to lie supine for a prolonged period of time. By preparing the patient for the conditions required you can minimise disruption to the surgery and assess the likelihood of compliance. Patients with restricted mobility, neurological movement disorders, dementia, learning difficulties or children may require general anaesthetic. If there are any concerns then the surgeon should be involved in the decision-making process.

Describe important points for consideration of intraoperative care of the patient under regional anaesthesia.

The patient should be made comfortable, pressure areas should be protected and temperature managed to ensure the comfort of the patient. Standard ECG, blood pressure and oxygen saturation monitoring should be applied. A right-angled bar can be used to allow tenting of drapes over the patients face and supplemental oxygen can be administered either using nasal cannula or a facemask. Patients with anxiety can be reassured by hand-holding, verbal reassurance and adjustment to surgical drapes. If required, anxiolytics and sedative medications can be administered in small titrated boluses e.g. 0.5 mg midazolam IV boluses, but avoid over-sedation of patients. If sedation is used then capnography should be monitored.

What are the local anaesthetic techniques used in eye surgery?

The nomenclature of the blocks is based on the anatomical location of the needle tip. There are three types of regional anaesthesia technique for ophthalmic surgery:

- Sub-Tenon's block: local anaesthetic is injected under the Tenon's capsule.
- Intra-conal (Retrobulbar) block: the needle tip is advanced to enter the conal space and local anaesthetic is injected into the muscle cone behind the globe.

- Extra-conal (peribulbar) block: The needle tip is advanced along the floor of the orbit and remains outside the cone.

The technique chosen depends on the patients' wishes, the type of surgery and the skill of the anaesthetist. Retrobulbar blocks have the highest risk of complications and so are commonly being replaced by Sub-Tenon's or peribulbar techniques. Sub-Tenon's blocks are the most popular choice due to reduction in the incidence of complications. It is still, however, associated with chemosis and subconjunctival haemorrhage and is contraindicated in patients who have undergone previous scleral banding and detachment surgery.

What are the contraindications to eye blocks?

These can be broken down into absolute and relative contraindications.

Absolute contraindications to regional anaesthesia of the eye include patient refusal, allergy to local anaesthetic, localised infection.

Relative contraindications include patients who are unable to lie still (e.g. movement disorders, uncontrolled cough), inability to follow commands (e.g. due to age or communication difficulties), high INR or abnormal coagulation and perforated globe or trauma.

How do you perform a Sub-Tenon's block?

As for all regional techniques, it is good practice to have a general introduction that includes the pre-op assessment of the patient, identification of contraindications, obtaining consent, checking the side of surgery and preparation of equipment and drugs.

I would start by inserting an IV cannula and ensuring adequate monitoring of the patient. I would obtain surface anaesthesia of the eye by applying a few drops of local anaesthetic, such as oxybuprocaine 0.4% or proxymetacaine 0.5% to the medial or lateral aspects of the eye. I would also clean the eye after it has been topically anaesthetised using povidone-iodine 5% aqueous solution drops to the conjunctiva. The patients can blink a couple of times to encourage spread to the whole eye surface. I would then apply a speculum to retract the eyelids and ask the patient to look upwards and outwards. Avoiding any conjunctival vessels, I would use non-toothed forceps to take a small bite of conjunctiva and Tenon's capsule in the inferonasal quadrant and a small incision is made through these layers using Westcott scissors. Through this opening I would pass a 19G curved, 25 mm Sub-Tenon's cannula with a 10 ml syringe containing local anaesthetic (usually 2% lidocaine with or without adrenaline or 0.5% bupivacaine can be used. Hyaluronidase can also be added.). The needle should be passed following the contour of the eyeball until the syringe is vertical. Aspiration should be attempted before slow injection of 3–5 ml of the local anaesthetic. I would then remove the needle and close the eye gently.

How would you perform a peribulbar block?

You can use the same introduction as for the Sub-Tenon's block, including the application of topical anaesthetic to the eye surface and ensuring asepsis.

In this technique, I would keep the globe in a neutral position by asking the patient to focus on a spot on the ceiling directly in front of them. I would pass a needle inferotemporally, lateral to the lateral limbus either through the limbus or through the skin.

I would advance my needle, keeping the direction of the needle parallel to the floor of the orbit, with the aim of keeping the needle tip in the inferotemporal quadrant of the orbit. I would then aspirate and inject 5–10 ml of local anaesthetic. I may be able to supplement this block with an additional medial peribulbar injection if required.

What are the complications of regional anaesthesia of the eye?

This answer can again be broken down into orbital and systemic complications, or mild and severe complications.

Complications of blocks may either be due to the technique and equipment used, or the local anaesthetic agents. With all anaesthetic complications, the surgeon performing the procedure should be informed and an explanation provided to the patient when appropriate. Orbital complications of ophthalmic regional blocks include failure of the block, wrong site of block, corneal abrasion, globe damage, optic nerve damage, chemosis and subconjunctival haemorrhage. Orbital haemorrhage is a sight-threatening complication of blocks. It can be the result of either venous or arterial bleeding and it can be concealed or revealed. Severe venous haemorrhage usually produces markedly blood-stained chemosis and slightly raised intraocular pressure. This can be managed by direct digital pressure as a tamponade effect. Arterial bleeding is often rapid, causes proptosis and reduced visual acuity and can raise intraocular pressure rapidly. Again, firm digital pressure can be applied although consideration must be given to the need for medical management such as acetazolamide or mannitol, or surgical management, such as lateral canthotomy or orbital decompression.

Systemic complications of regional anaesthesia of the eye include allergic reaction, local anaesthetic toxicity, oculocardic reflex and brainstem anaesthesia. Central spread of local anaesthetic can occur if the tip of the needle enters the optic nerve sheath. This sheath can act as a conduit for local anaesthetic to pass to the brain. Symptoms the patient can experience include drowsiness, vomiting, contralateral palsy due to optic chiasm involvement, seizures and cardiorespiratory arrest. These symptoms usually appear rapidly and treatment is supportive.

Further Reading

Anker R, Kaur N. Regional anaesthesia for ophthalmic surgery. *BJA Education*. 2017;17(7):221–227.

Royal College of Anaesthetists. The Guidelines for the Provision of Anaesthetic Services (GPAS). Chapter 13: Guidelines for the

Provision of Ophthalmic Anaesthesia Services 2022.

Smith G, Aitkenhead AR. Smith and Aitkenhead's Textbook of Anaesthesia. 7th ed. Thompson J, Moppet I, Wiles M, editors: Elsevier; 2019.

2.8.1 Procedures under Tourniquet, Reperfusion Injury and Antioxidants – Matthew J Aldridge

You are asked to anaesthetise for an elective orthopaedic list. Your first patient is a 78-year-old woman for a right total knee replacement.

What are the primary issues you would want to consider?

As you have not been given much in the way of specific information for this patient, give a brief list of the likely main concerns in a case such as this, then move on to the rest of your normal anaesthetic history.

This is an elderly woman who is to undergo major elective orthopaedic surgery. My main concerns would be that of significant comorbidities, possibly masked by pain-related reduced exercise tolerance and also the potential effects of undergoing joint replacement surgery. These would include blood loss and/or tourniquet and cement use, fluid balance and adequate pain and temperature control.

As part of my anaesthetic history, I would enquire as to previous anaesthetics, as she may have had an arthritic joint replaced before, also about illnesses and medications, allergies and smoking, alcohol intake, reflux and fasting status. Airway assessment should be conducted with history and examination and there may be useful information in old anaesthetic charts. The presence of rheumatoid arthritis would also be significant because of the risk of reduced neck and temporomandibular joint mobility.

Specifically, I would seek evidence of cardiorespiratory disease, bearing in mind that she may have asymptomatic ischaemic heart disease if she cannot walk far. I would also seek contraindications to planned surgical or anaesthetic techniques. Examples of this include neuraxial or peripheral nerve blockade and anticoagulation medication, or tourniquet use, sickle cell anaemia or profound peripheral vascular disease.

Another major concern after this type of procedure can be the risk of deep venous thrombosis and pulmonary embolism so a previous history of this or additional risk factors should be sought.

Your surgeon tells you he routinely uses a tourniquet for knee replacements. In your experience, in which situations are tourniquets generally used?

Limb tourniquets are used to reduce bleeding and improve operating conditions in mainly orthopaedic or plastic surgery but are also used in intravenous regional

anaesthesia for procedures such as manipulation of Colles fractures. As the tourniquet occludes the blood supply to the limb, the use is limited by time.

What general concerns might you have with tourniquet use?

These could be divided into preoperative, intraoperative and postoperative.

Preoperative considerations include sickle cell disease, poorly controlled or unstable cardiac failure, critical peripheral vascular ischaemia and the presence of deep venous thrombosis that could be dislodged to become pulmonary emboli. Patients with uncontrolled hypertension may have exaggerated responses to tourniquet-related hemodynamic changes.

Intraoperatively, the application of the tourniquet requires limb exsanguination which could precipitate ventricular overload and pulmonary oedema. There may be a hypertensive response to prolonged tourniquet inflation even under general anaesthesia. A dull aching tourniquet pain may be felt if the patient is awake, despite peripheral blockade. The total tourniquet inflation time should be limited to an absolute maximum of 2 hours to prevent permanent ischaemic damage.

Postoperatively, there is a haemodynamic and metabolic response when the tourniquet is deflated at the end of surgery and this, coupled with blood loss, may cause profound hypotension in the elderly. The metabolic insult from the ischaemic limb may worsen already poor cardiac or renal function. Of most concern is the rare risk of irreversible ischaemia and limb loss. In very high-risk patients it may be appropriate to consider surgery without using a tourniquet and some surgeons routinely operate like this.

What physical injuries may result from tourniquet use?

This may form part of an answer to a more general question on physical injuries to patients during anaesthesia

The mechanism of injury here is tissue compression resulting in ischaemia. This can affect skin, nerves, muscle or blood vessels. Skin injuries include blistering and bruising of skin, or friction burns from loosely applied tourniquets. Tourniquets may also form a reservoir for antiseptic solution with potential for chemical injury. Nerve injuries most commonly affect the radial and sciatic nerves, and are more common with excessive inflation times or pressures.

How is the tourniquet inflated?

Just describe what happens in theatre.

Before the tourniquet is inflated it is padded and the limb has to be exsanguinated by elevation or application of a rubber Esmarch bandage. This is not done in the presence of infection or tumour. The skin beneath the cuff should be well padded and free of folds, and the distal edge sealed to prevent leakage of antiseptic under the tourniquet.

Tourniquets should be inflated to a pressure of 40–80 mmHg above the systolic blood pressure, indicated by the point at which a distal pulse is lost. After 120 minutes the tourniquet should be deflated for at least 10 minutes to allow reperfusion.

What are the physiological effects of tourniquet inflation?

Exsanguination and inflation expands the central venous blood volume and increases the peripheral vascular resistance which causes a rise in central venous and arterial pressure.

A rise in pulmonary artery pressure can be seen in people with extensive varicose veins or poor ventricular compliance. There is a temperature reduction in the non-perfused limb and the production of anaerobic metabolites after just a few minutes. From about an hour after inflation there is a rising blood pressure and a tachycardia that is usually resistant to treatment such as deepening of anaesthesia. This may be because of cellular ischaemia so vasodilators or clonidine can sometimes moderate the response.

What is tourniquet pain?

This can be felt by patients as an aching or burning pain in the distal limb about an hour after tourniquet inflation. Intravenous analgesia does not always work and the only really effective method is to deflate the tourniquet temporarily.

How can tourniquet pain be managed if the patient is having awake regional anaesthesia on the upper limb?

Tourniquet pain may be experienced by any patient undergoing awake upper limb surgery with a tourniquet regardless of choice of regional technique. Although the axillary approach to blocking the brachial plexus is associated with reduced complications, tourniquets are often less well tolerated than with the supra or infraclavicular approach.

In addition to a brachial plexus block, the intercostobrachial nerve can be anaesthetised by depositing 5 ml of local anaesthetic along the axillary crease. This may reduce the incidence of tourniquet pain and is a useful supplement to any brachial plexus block. However, this will not block the ischaemic component of tourniquet pain, and temporary deflation of the tourniquet may still be necessary.

Returning to your patient for knee replacement, you choose to perform a spinal block with sedation. Things proceed well but it becomes a teaching case for the new orthopaedic registrar so surgery is prolonged. Shortly after the tourniquet is deflated and the sedation turned off, the patient becomes distressed and tachypnoeic.

What could be happening?

Consider differential diagnoses as well as relating your information to this specific case.

The respiratory distress is likely to be related to the release of the tourniquet given the timing. It may be that she has an exacerbation of a pre-existing condition such as cardiac failure or precipitation of a myocardial ischaemic event which could be contributed to by reperfusion effects. It could also be an embolic event such as a fat or pulmonary embolus. It is possible that a reaction to the cement has been delayed by having had a tourniquet inflated.

What do you know about tourniquet release and reperfusion syndrome?

Reperfusion injury syndrome is a paradoxical tissue injury after blood flow is restored after a period of ischaemia. Deflation leads to the limb being reperfused and so reversal of the effects seen with inflation. Therefore, there is a drop in systemic vascular resistance, blood pressure, central venous pressure and heart rate. This hypotension is exacerbated by vasodilatation secondary to the anaerobic metabolites and increased carbon dioxide from the limb. End-tidal CO₂ increases, there is a reduced oxygen tension in blood from the limb and subsequently an increased oxygen consumption. The patient's

core temperature falls and they develop a mixed acidosis. There is also an increase in intracellular calcium and extracellular potassium, cell swelling and free radicals and the limb becomes red which is due to a reactive hyperperfusion.

Hypoxia and hypotension may be possibly worsened by delayed cement implantation effects.

Does this matter?

Fit and healthy patients can tolerate this well but in older patients with poor cardiopulmonary reserve there may be a depressant effect on myocardial contractility, producing ischaemia, hypoxia and hypotension.

Can the reperfusion effects be reduced or prevented?

This can be answered with common sense.

Use of a regional technique, with or without a general anaesthesia, may ease the metabolic effects. The use of total intravenous anaesthesia rather than an inhalational anaesthetic might scavenge free radicals. Limiting the time that the tourniquet is inflated and using limits for inflation pressures should help minimise anaerobic metabolism. Maintaining physiological normality as much as possible (for instance blood pressure, temperature and hydration) puts the patient in the best position to recover. Hyperventilation, if ventilation is being controlled, can be used after tourniquet release to reduce end-tidal CO_2 and acidosis.

Do you know anything about free radicals and antioxidants in this situation?

You could extrapolate information from reperfusion injury to other tissues, e.g., myocardial injury.

Free radicals are produced as part of reperfusion injury. They are molecules with unpaired electrons and act as intermediaries in biological reactions, reducing oxygen to oxide ions. They are normally part of the body's defence system but in ischaemia the production overwhelms normal controls and causes cell damage such as oxidation of cell membrane lipids.

The free radicals are usually controlled by antioxidants like vitamin E or glutathione, which act as scavengers, or by superoxide dismutase. It has been suggested that administration of antioxidants may help to limit tissue injury in ischaemia, but this has only been demonstrated in experimental models.

Are there any specific concerns to consider once your patient has been moved to the recovery room?

Relate these to the case to answer the question but also include relevant general post-operative considerations.

Use of a tourniquet during surgery could lead to delayed reperfusion effects including hypotension occurring after the patient has been moved to recovery. Another particular concern would be that of blood loss which may begin once the tourniquet has been deflated.

Other concerns relate to temperature control and pain management. Patients often get cold in orthopaedic theatres despite the use of warming devices. Patients' temperature must be monitored regularly and forced-air warmers and fluid warmers used for all operations lasting longer than 30 minutes. Patients must be actively warmed in recovery if their temperature is less than 36°C, to avoid increased oxygen consumption from shivering and coagulation abnormalities.

Pain must also be well managed; however, in this case the spinal block should still be in effect. Appropriate analgesia should still be prescribed for use once this has worn off.

Deloughry JL, Griffiths R. Arterial tourniquets. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2009; 9(2): 56–60.

Kahraman S, Kilinc K, Dal D, Erdem K. Propofol attenuates formation of lipid peroxides in tourniquet-induced ischaemia-reperfusion injury. *British Journal of Anaesthesia*. 1997; 78(3): 279–281.

Orban JC, Levraut J, Gindre S, et al. Effects of acetylcysteine and ischaemic preconditioning on muscular function and postoperative pain after orthopaedic surgery using a pneumatic tourniquet. *European Journal of Anaesthesiology*. 2006; 23(12): 1025–1030.

2.8.2 Major Trauma, Hospital Triage and Scoring Systems – Anandh Balu

Can you define major trauma?

Major trauma refers to significant injury or multiple injuries that are potentially life-threatening or changing. An alternative definition is an injury severity score (ISS) greater than 15. Major trauma is the leading cause of death in patients under the age of 45 and can lead to long-term disabilities or lengthy rehabilitation with complete or incomplete functional recovery.

How are patients triaged?

Patients are usually triaged by the attending paramedic crew or pre-hospital team unless a major incident has been declared. This can be done using various pre-hospital triage tools which may differ from region to region but generally these tools take into account: physiology and vital signs, anatomical site of injury as well as those with underlying or complex health needs. Practitioners will alert the trauma desk to severely injured patients.

The trauma desk team can provide advice to the pre-hospital team but can also generate alerts and abbreviated handovers with live updates for the receiving trauma team. Pre-alert information should include: age and sex of the patient, time of incident, mechanism of injury, injuries suspected, vital signs, treatment so far, estimated time of arrival and special requirements. Also included will be the ambulance call sign with both the name of the person taking the call and the time of the call.

The preferred destination for any major trauma patient is usually a major trauma centre (MTC); however, if transfer cannot be achieved safely within 45 minutes from the

scene then the patient may need to be moved to the nearest trauma unit (if closer) for life-saving interventions and initial resuscitation and assessment prior to being moved onwards to an MTC.

Describe the organisation of the trauma network.

Following numerous reviews and reports, care of the trauma patient in the UK has been restructured over the last two decades to form regional trauma units and major trauma centres. A trauma unit will be able to provide the personnel and resources for initial stabilisation of a patient who is too far away from a major trauma centre. A major trauma centre will have the full multidisciplinary team and resources to provide definitive care for a severely injured patient and will have significant experience due to high volume of exposure – at least 650 cases per year. Additionally, an MTC will have a structure in place for critical care, ward-based care and ongoing specialist trauma rehabilitation. Trauma Audit and Research Network (TARN) has demonstrated consistently improving patient outcomes since its inception and focusses on collection and reporting of patient outcomes as well as service improvements and innovation.

There is now increased recognition of the impact of lower energy mechanisms of injury in elderly patients who may be frail, have multiple comorbidities or are anticoagulated. TARN has highlighted that elderly patients commonly have severe injuries with high mortality rates from head injuries or falling from standing. This has led to the advent of silver trauma calls involving elderly patients; the aim is to deliver multidisciplinary patient-centred care from arrival.

Paediatric trauma care is organised differently from region to region depending on the hospitals within the region but follows a similar theme.

Tell me about scoring systems in major trauma that you are aware of and their clinical uses.

Scoring systems in major trauma may be based on physiological variables or anatomical injuries or a combination. Commonly used examples include: revised trauma score (RTS), injury severity score (ISS) and the trauma and injury severity score (TRISS). RTS is a physiological scoring system derived from a combination of Glasgow Coma Score (GCS), respiratory rate (RR) and systolic blood pressure (SBP). The ISS is an anatomical scoring system derived from the abbreviated injury scale (AIS) and examines six body regions. The AIS grades injuries from 1 to 6 with 1 being minor and 6 being unsurvivable. The ISS is derived from the sum of the squares of the highest AIS in the three most injured body regions and ranges from 1 to 75. An AIS of 6 corresponds to an ISS of 75. TRISS is a scoring system which utilises a combination of the ISS and RTS as well as the patient age and type of injury. All of these systems have their limitations. TRISS has been used to estimate probability of survival and ISS has been shown to correlate with mortality, morbidity and length of inpatient stay. High ISS scores are a validated predictor of post-injury multiorgan failure.

What is the role of the anaesthetist in receiving major trauma patients?

Anaesthetic representation is a key part of the trauma team. Typically, the trauma team leader will be an emergency department physician. The remainder of the team will usually consist of:

general surgeon, orthopaedic surgeon and in some departments an intensive care physician, neurosurgeon and radiologist. The anaesthetist usually takes a non-leadership role due to a combination of task-orientated roles in initial airway management with concurrent resuscitation. Securing the airway can be challenging in trauma patients due to distorted anatomy, cervical spine stabilisation and shocked state with extreme physiological derangement. NAP4 showed that there was a higher incidence of difficult airway in trauma intubations due to a variety of factors amongst which included injury-related patient factors as well as being in a high pressure, time-critical situation. Invasive monitoring and large bore venous or central venous access is required but should occur in parallel with other assessment and treatment. Being at the head end puts the anaesthetist in a unique position of being able to make a holistic assessment of the situation. This can lead to sharing of information and mental models with the team leader and the rest of the team. The responsible anaesthetist will often provide continuity and ongoing care for the patient through their initial journey whether this is via diagnostic imaging such as a trauma-series CT, interventional radiography, intensive care or theatre for damage control surgery. Consultant anaesthetists often work as part of the major trauma service and can be responsible for coordinating care of the severely/multiply injured patient.

NICE Guideline NG39; Major trauma: assessment and initial management. 2022 [cited 30 May 2022]. Available from: www.nice.org.uk/guidance/ng39/evidence/full-guideline-2308122833

McCullough A, Haycock J, Forward D, Moran C. Early management of the severely injured major trauma patient. *British Journal of Anaesthesia*. 2014;113(2):234–241.

Nickson C. Trauma Scoring Systems. Life in the Fast Lane • LITFL. 2022 [cited

30 May 2022]. Available from: <https://litfl.com/trauma-scoring-systems/>.

Sengupta S, Shirley P. Trauma anaesthesia and critical care: The post trauma network era. *Continuing Education in Anaesthesia Critical Care and Pain*. 2014;14(1):32–37.

Silver Trauma – RCEMLearning [Internet]. RCEMLearning. 2022 [cited 30 May 2022]. Available from: www.rcemlearning.co.uk/foamed/silver-trauma/.

2.8.3 Blood Conservation Strategies – Niladri Das

How do you define anaemia?

Anaemia is a condition whereby the number of red blood cells and their oxygen-carrying capacity are unable to meet the body's physiological demand. It is quantified by a haemoglobin concentration of less than 130 g/L for men and less than 120 g/L for non-pregnant women. Anaemia is typically categorised according to the mean cell volume (MCV) and hence defined as microcytic (MCV <80 fl), macrocytic (MCV >96 fl) or normocytic (MCV 80–96 fl). Perioperative anaemia is an independent risk factor for increased morbidity and mortality in addition to a strong predictor for perioperative blood transfusion requirements.

What do you understand by the term patient blood management?

Patient blood management involves a multidisciplinary, evidence-based approach to management of perioperative anaemia and blood transfusion in order to improve patient

outcomes. The clinical concept focusses on optimisation of red cell mass preoperatively, minimisation of blood loss and management of postoperative anaemia.

A 65-year-old man is listed for abdominoperineal resection surgery. His haemoglobin level is 95 g/L. What are the options for preoperative optimisation of his anaemia?

All patients should have a full blood count evaluated at least 4–6 weeks prior to major elective surgery. Patients who are found to have low haemoglobin levels should have haematinics measured to aid diagnosis and subsequently treat the cause of anaemia.

Iron deficiency anaemia can be treated with oral iron preparations although compliance may be poor due to gastrointestinal side effects. Patients with an ongoing inflammatory process or malignancy can have inadequate enteral absorption. Oral iron therapy has been shown to reduce blood transfusion requirements; however, a period of 3 months or more is often required to significantly improve iron stores within the body which of course is a limitation for more urgent surgery. Alternatively, intravenous iron preparations have been shown to be effective with an improved side effect and safety profile compared to oral iron. Iron infusions should be administered in an appropriate environment with facilities available to monitor patients and treat any complications. The synthesis of red blood cells, known as erythropoiesis, is regulated by the hormone erythropoietin (EPO) which is secreted by the kidneys. Synthetic recombinant EPO has been used in patients with chronic kidney disease on dialysis to stimulate proliferation of red blood cells. However, due to the increased risk of thrombotic complications and mortality, EPO is not recommended in the surgical population to reduce the need for blood transfusions perioperatively.

What strategies are available to minimise perioperative bleeding?

Preoperative risk stratification is of key importance. The type of surgical intervention should be considered with respect to bleeding risk. A thorough preoperative assessment should be undertaken to elicit a history of bleeding diathesis as well as use of anticoagulants and antiplatelet agents. An individual assessment of the thrombotic risk of suspending these drugs against the risk of perioperative bleeding must be made. Patients with drug-eluting coronary stents are at high risk of stent thrombosis and myocardial infarction and should continue their dual antiplatelet agents into the perioperative period. Bridging of long-acting anticoagulants, such as warfarin, may be necessary and referral to a haematologist can be made to provide a plan for perioperative anticoagulation. Novel anticoagulants, such as rivaroxaban and dabigatran, have a more predictable pharmacokinetic profile and require preoperative discontinuation unless there is a low risk of surgical bleeding.

Intraoperative blood loss may be influenced by surgical technique, anaesthetic blood loss reduction strategies and pharmacological management. Minimally invasive surgery, to include laparoscopic and endovascular techniques, are associated with reduced blood loss when compared to open procedures. Meticulous surgical haemostasis is important when preventing perioperative bleeding. Anaesthetic blood conservation strategies

include use of regional anaesthesia, particularly in major orthopaedic surgery, and maintenance of a balanced physiology. Optimum conditions for clot formation can be provided through avoiding hypothermia, acidosis and hypocalcaemia. Although manipulation of cardiovascular physiology can improve the surgical field and reduce blood loss, hypotensive anaesthesia remains a contentious subject matter. Point-of-care testing, for example thromboelastography, is increasingly being used to guide blood product administration and management of coagulopathy. Cell salvage is recommended in cases where blood loss is greater than 500 ml in addition to antifibrinolytic drugs, such as tranexamic acid, which should be used prophylactically in major surgery where blood loss is expected to be high. The clinical randomisation of an antifibrinolytic in significant haemorrhage (CRASH-2 trial) demonstrated the benefit of early therapy with tranexamic acid in trauma-induced bleeding. Measures to reduce bleeding, avoid coagulopathy and minimise blood loss should be continued into the postoperative period in order to reduce the incidence of anaemia. The use of postoperative drains can be minimised and is often a part of the enhanced recovery after surgery protocol. The frequency of phlebotomy and volume of blood sample obtained for tests can be reduced. It is also important to note that the surgical stress response can precipitate a functional iron deficiency state and consideration should be given to replenishing body iron stores through administration of intravenous iron in the postoperative period.

What are the principles of cell salvage?

Cell salvage, a method of autologous blood transfusion, is an essential component of patient blood management and effective blood conservation strategy. The process involves the collection of shed blood from the operative field which is then mixed with an anticoagulant as it is aspirated using a suction device into a collection reservoir. The blood passes through a filter while separation of red cells from the anticoagulated blood occurs through centrifugation. The red cells are finally washed using 0.9% saline and pumped into a bag ready for the patient. Re-infusion of salvaged blood should be completed within 4 hours after the processing for intraoperative cell salvage and 6 hours after the start of collection for postoperative cell salvage. Routine bedside checks should be performed prior to transfusion in the same manner as for allogeneic blood. It is recommended that cell salvage equipment and trained staff should be available 24 hours a day in hospitals undertaking major surgery where blood loss is a recognised complication.

Cell salvage, alongside other patient blood management measures, should be considered for all surgical procedures where blood loss is estimated to exceed 500 ml in adult patients or greater than 8 ml/kg in children. It is commonly used in cardiac, vascular, obstetric, major urological and orthopaedic surgery. In cases where there is doubt over the expected blood loss, the device can be set up to 'collect only'. It should also be considered in patients with anaemia requiring urgent surgery, patients with known coagulopathy or a religious objection to receiving an allogeneic transfusion. There are no absolute contraindications to cell salvage. Patient refusal is uncommon and most Jehovah's Witness patients will accept cell salvage. Contamination of the aspirated blood with bowel contents, infection or tumour cells is considered a relative contraindication and a risk versus benefit assessment should be made in such cases; this should be discussed with the patient and consent appropriately documented prior to surgery.

What are the beliefs of a Jehovah's Witness?

The fundamental principle behind the faith of a Jehovah's Witness is that all life is sacred, it is given by God, belongs to God and He is the only one with the right to determine how it should be used. The Bible teaches that sacred life is represented by the blood of a creature and therefore taking the blood of another creature, either enterally or parentally, violates the law of the Bible. It is a core value that is followed even in the face of clinical advice that recommends an allogeneic blood transfusion to maintain life.

How will you manage a patient that has refused a blood transfusion?

It is important to note that not all patients who refuse a blood transfusion are Jehovah's Witnesses and not all Jehovah's Witnesses refuse blood transfusion. It is imperative that all individuals be given a clear explanation of the procedures that may require blood transfusion perioperatively, the risks involved in refusal of these treatments and alternative options. A Jehovah's Witness patient should be given the opportunity to discuss this with their family, friends or a member of the Jehovah's Witness Hospital Liaison Committee. Ultimately, adults with capacity have the right to decide which treatments to accept or refuse and this must be respected regardless of reason. In cases where an individual has lost the capacity to consent, clinicians should pursue whether a valid and applicable advanced decision has been made.

A Jehovah's Witness patient who follows the teaching of their church in relation to blood transfusion will not accept whole blood or its derivatives to include red blood cells, fresh frozen plasma and platelets. However, patients may accept, as a matter of individual choice, products such as cryoprecipitate, fibrinogen concentrate, prothrombin complex concentrate and human albumin solution. Jehovah's Witnesses also accept recombinant coagulation factors and drugs such as iron and erythropoietin which are not derived from blood. Preoperative autologous blood donation and acute normovolaemic haemodilution is again a matter of individual choice. Procedures such as cell salvage, renal replacement therapy, cardiopulmonary bypass and extracorporeal membrane oxygenation are usually acceptable. Clear documentation in the medical records must be made to which treatments the patient consents and those to which they do not consent.

In order to facilitate planning, a preoperative multidisciplinary meeting is advised to include a senior surgeon, anaesthetist and haematologist. At least 6 weeks prior to major elective surgery with an expected significant blood loss, the patient's haemoglobin level should be evaluated and optimised with iron and erythropoietin. On the day of surgery, any relevant issues should be discussed at team brief and a specific record of which blood products/procedures the patient will or will not accept be made available at the time of the WHO Surgical Safety checks in theatre. The interventions recommended as part of blood conservation strategies should be applied perioperatively as discussed before. A thorough verbal and written handover of events is essential following surgery.

Further Reading

Thakrar SV, Clevenger B, Mallet S. Patient blood management and perioperative anaemia, *BJA Education*. 2017, 17: 28–34.

Klein A, Bailey C, Charlton A et al. Cell salvage for peri-operative blood conservation.

Association of Anaesthetists. September 2018.

Klein A, Bailey C, Charlton A. Anaesthesia and peri-operative care for Jehovah's Witnesses and patients who refuse blood, *Association of Anaesthetists*. July 2018.

2.8.4 Bone Cement Implantation Syndrome – Joseph Swani

What is bone cement implantation syndrome (BCIS)?

Bone cement implantation syndrome (BCIS) is a potentially fatal complication which can occur in patients undergoing cemented bone surgery. It has been described in a variety of procedures involving pressurised bone cement but is most commonly associated with cemented hip arthroplasty. It is usually seen at the time of bone cementation and prosthesis insertion, but can also occur before cementation during preparation of the femoral canal, or after cementation when the joint is reduced or limb tourniquet deflated.

What is the composition of bone cement?

Bone cement consists of a powder and a liquid which are mixed together to form the cement. The powder contains pre-polymerised polymethyl methacrylate (PMMA) and the main liquid component is the liquid monomer of methyl methacrylate (MMA). The powder and liquid are mixed together in theatre with a catalyst, resulting in polymerisation of the monomer fluid. As the reaction continues the viscosity of the mixture increases over time with eventual hardening of the cement. Antibiotics can also be incorporated into the mixture.

Why is cement used and what are the benefits of cemented vs uncemented prostheses for hip fracture surgery?

The cement acts as a grout, filling the gaps between the metal prosthesis and bone. This results in a more secure prosthesis and a higher likelihood of pain-free mobility after surgery. There is also a reduced need for future revision surgery as there is less risk of loosening of the prosthesis.

What are the clinical features of BCIS and how it can be classified?

Clinically there are a number of possible features. Hypoxia may be recognised by a fall in arterial oxygen saturations. In the awake patient signs include breathlessness and reduced conscious level. Systemic hypotension can occur, as well as arrhythmias. An increase in pulmonary vascular resistance occurs, a fall in end-tidal CO₂ can indicate right heart failure and/or a significant drop in cardiac output. Severe cardiovascular collapse can occur leading to cardiac arrest.

BCIS can be classified into three grades:

- Grade one is where there is a fall in arterial oxygen saturation below 94% and/or a decrease in systolic blood pressure by more than 20%.
- Grade two is where there is a fall in arterial oxygen saturation below 88% and/or a decrease in systolic blood pressure by more than 40%, or if there is loss of consciousness.
- Grade three is when cardiopulmonary resuscitation is required.

What patient factors are associated with an increased risk of BCIS?

These include increasing age, significant cardiopulmonary disease, ASA grade 3 or 4, male sex and medication with diuretics or warfarin.

Osteoporosis, bony metastases and hip fractures (particularly pathological or intertrochanteric fractures) also increase risk, possibly due to abnormal or increased vascular channels through which marrow contents can get into the circulation.

What is the pathophysiology?

It is not fully understood; however, there are several proposed models.

One of the components of bone cement is MMA. The initial monomer-mediated model theorised that circulating MMA monomers led to hypotension after it was shown that they can cause vasodilatation in vitro. However, the plasma concentrations of MMA were shown to be much lower than that needed to cause these cardiovascular effects in vivo.

The embolic model describes how material including bone particles, fat, cement, marrow, air and aggregates of platelets and fibrin embolise from the area of cementation to the right atrium, right ventricle and pulmonary artery. The emboli may promote the release of mediators as well as causing mechanical obstruction. Mediator-induced pulmonary vasoconstriction, in addition to mechanical obstruction from the emboli, causes an increase in pulmonary vascular resistance and also V/Q mismatch leading to hypoxaemia. It is thought that increased pulmonary vascular resistance causes a reduction in right ventricular ejection fraction. The thin-walled and compliant right ventricle then distends, which shifts the interventricular septum into the left ventricle, further reducing left ventricular filling and causing a drop in cardiac output. Other mediators may cause a reduction in systemic vascular resistance, further contributing to systemic hypotension.

However, embolisation does not always cause BCIS features and it has been demonstrated that the extent of embolisation is not proportional to the degree of hypotension. It is likely that other mechanisms also contribute to the features seen. These may include histamine release, either as an IgE-mediated process or a direct effect of cement monomer, as well as complement activation.

How can theatre teams prepare for the possibility of BCIS?

Patients who are of higher risk should be identified prior to surgery. Members of the theatre team should be aware of the potential for BCIS to occur with femoral instrumentation and the use of cemented prostheses and this should be discussed for each patient as part of the pre-list briefing, as well as the World Health Organisation (WHO) Surgical Safety Checklist 'time-out' before skin incision. Some hospitals use a 'cement curfew', this includes assigning roles to theatre team members before surgery commences. All theatre team members with assigned roles are to be present in theatre at the time of prosthesis insertion with increased vigilance from these members during this time. If evidence of a severe reaction or cardiopulmonary arrest occurs then these members can perform their pre-defined roles, improving the efficiency and organisation of the resuscitation process.

What specific intraoperative steps can the surgeon and anaesthetist take to reduce the risk of BCIS?

For the surgeon:

- Inform the anaesthetist when cementing is about to occur
- Thoroughly wash and dry the femoral canal

- Apply cement in a retrograde fashion using the cement gun with a suction catheter and intramedullary plug in the femoral shaft
- Avoid vigorous pressurisation of cement in patients judged to be high risk.

For the anaesthetist:

- Ensure adequate hydration pre- and intraoperatively
- When informed by the surgical team that cementing is about to occur, confirm with them that this has been heard
- Maintain vigilance for signs of cardiorespiratory compromise around the time of cementing
- Aim to keep systolic blood pressure within 20% of pre-induction value throughout surgery using vasopressors and/or fluids
- Have vasopressors prepared in case of cardiovascular collapse.

Is BCIS reversible?

BCIS is a reversible, time-limited phenomenon. Pulmonary artery pressures can normalise within 24 hours and may only take a few minutes to recover in some patients. Prompt recognition and initiation of supportive treatment is vital to improve outcome in this potentially life-threatening condition.

What is the management of BCIS?

The management of BCIS is supportive with attempts to normalise oxygen levels, improve myocardial contractility and normalise blood pressure. It requires clear communication between the surgical and anaesthetic members of the theatre team.

100% oxygen should be applied and the airway secured if necessary.

The management of cardiovascular collapse should be in line with the principles of treating pulmonary artery hypertension and right ventricular failure. Intravenous fluids are used to optimise preload. Peripherally administered vasopressors are often given first line for systemic hypotension, they primarily increase systemic vascular resistance and maintain systemic blood pressure, improving coronary artery perfusion and thus myocardial contractility. Central venous catheter insertion may be indicated when simple measures fail; this allows the administration of central vasopressors and inotropic agents. Inotropes, including dobutamine and milrinone, can be used to improve right ventricular contractility. Cardiac output monitoring should be considered, to help guide therapy with intravenous fluids, vasopressors and inotropes. Pulmonary vasodilators may be needed to reduce pulmonary artery pressures. If cardiac arrest occurs, then management should be as per the advanced life support algorithm.

Postoperatively, the patient should be managed in the intensive care unit.

Further Reading

Donaldson J, Thomson HE, Harper NJ, et al. Bone cement implantation syndrome. *British Journal of Anaesthesia*. 2009; 102: 12–22.

Griffiths R, Parker M. Bone cement implantation syndrome and proximal

femoral fracture. *British Journal of Anaesthesia*. 2015; 114: 6–7.

Griffiths R, White SM, Moppett IK, et al. Safety guideline: Reducing the risk from cemented hemiarthroplasty for hip fracture 2015: Association of Anaesthetists of Great Britain and Ireland British Orthopaedic

Association British Geriatric Society. *Anaesthesia* 2015; 70: 623–626.

Khanna G, Cernovsky J. Bone cement and the implications for anaesthesia. *Continuing Education in Anaesthesia and Critical Care and Pain*. 2012; 12: 213–216.

Olsen F, Kotyra M, Houltz E, et al. Bone cement implantation syndrome in cemented hemiarthroplasty for femoral neck fracture: Incidence, risk factors, and

effect on outcome. *British Journal of Anaesthesia* 2014; 113: 800–806.

Shelton C, White S. Anaesthesia for hip fracture repair. *British Journal of Anaesthesia*. 2020; 20: 142–149.

So D, Yu C, Doane M.A. Bone cement implantation syndrome. Anaesthesia tutorial of the week 2017. <https://resources.wfsahq.org/atotw/bone-cement-implantation-syndrome/>.

2.8.5 Management of Hip Fractures – Daniel El-Dalil

You are the anaesthetist on call for the trauma list, an 82-year-old man has been admitted following a fall and has suffered a right-sided neck of femur fracture. You have been asked to review him preoperatively, how would you approach this?

It is important to detail your routine preoperative anaesthetic assessment in a systematic manner while focussing on the specific considerations of anaesthetics in an elderly man.

I would start by taking a systematic history from the patient ensuring to consider the cause of the fall and if there was any syncope or pre-syncope symptoms. I would also enquire about their past medical history, previous anaesthetics, exercise tolerance, drug history and allergies. I would also like to know if there is any history of reflux and when the patient last had anything to eat or drink. I would then conduct a thorough examination, making sure to be vigilant for any other injuries, as well as assessing the patient's airway.

Elderly patients are likely to have underlying comorbidities, and specific focus should be on enquiring about any cardiovascular and respiratory illness, as this will help guide the choice of anaesthesia. I would also want to perform some investigations including a 12-lead ECG, and baseline blood tests including a clotting screen and group and save.

Hip fractures are associated with significant morbidity and mortality; I would assess the risk of mortality for the patient using the Nottingham Hip Fracture Score. I would also like to do a preoperative AMT-4 assessment as there is a high incidence of delirium associated with hip fractures and a baseline cognitive test will aid in diagnosis of postoperative delirium.

Despite patients with hip fractures often being high-risk surgical candidates, research has shown that outcomes with regards to mortality are better with surgery than with a non-operative approach. Timely surgery is important as it is associated with reduced pain, mortality, and rates of delirium. As such significant pre-optimisation is not feasible; however, I would want to correct any significant anaemia prior to surgery aiming for a Hb target of 90 g/L, as well as trying to avoid hypovolaemia by allowing the patient to drink clear fluids until 1 hour prior to the operation and considering the use of IV fluids.

To help aid preoperative analgesia I would perform a fascia iliaca block if there were no contraindications; further regional nerve blocks could be repeated at the time of surgery if more than 6 hours has elapsed.

There has been no clear evidence demonstrating reduced morbidity or mortality when comparing general vs spinal for hip fractures; therefore my decision would be based on the patient's comorbidities and preference.

Lastly it is important to note that there is a high mortality associated with hip fractures and as such I would like to have a discussion with the patient and their family about resuscitation and escalation status.

The patient states he tripped over a step while in the garden and he denied losing consciousness or of having any pre-syncopal symptoms. He was examined by the clinicians in ED and has no other injuries.

He has a past medical history of Type 2 diabetes, hypertension, and benign prostatic hyperplasia. He has no known drug allergies, and takes metformin 500 mg bd, amlodipine 10 mg and tamsulosin 400 mcg daily.

He has never smoked and lives alone in a bungalow, is independent of activities of daily living, and has an exercise tolerance of approximately 250 metres.

He last had something to eat and drink 8 hours ago. He has no preference regarding his choice of anaesthesia.

How would you perform the anaesthetic for this patient?

There has been no clear evidence to suggest one method is superior – it is therefore important to discuss the factors which will influence the anaesthetic approach.

My preference would be to perform a spinal anaesthetic for this patient as although he does not have any known cardiovascular disease, he may have undiagnosed ischaemic heart disease considering his age and risk factors such as hypertension and diabetes.

The ASAP-2 trial demonstrated that there was a significant increase in mortality in patients with intraoperative hypotension. I would therefore have non-invasive blood pressure monitoring on a 2.5 minute cycle and have a low threshold for invasive blood pressure monitoring. To avoid having a labile blood pressure, I would run a metaraminol infusion at a low level aiming for a MAP of >70 mmHg.

There is moderate quality evidence to suggest tranexamic acid reduces the need for intraoperative transfusion so I would give a stat dose of 15 mg/kg during induction.

If the patient was due to have a cemented prosthesis I would make a point of asking the surgeon to let me know prior to cementing so that I could be vigilant for bone cement implantation syndrome.

If the patient had not already had a fascia iliaca block or 6 hours had elapsed since they had their block, I would repeat this using a mixture of lidocaine 1% and levobupivacaine 0.25%.

With regards to the spinal anaesthesia, I would be wanting to use a relatively low dose of intrathecal bupivacaine: 7.5–10 mg, as well as considering giving 10–20 micrograms of intrathecal fentanyl. If the patient was unable to be positioned in a sitting position due to his pain, I would consider positioning him laterally with his injured hip facing upwards. I would want to ensure that there was good spread and density of the block and would test this with two sensory modalities.

Delirium is a common complication associated with hip fractures so to reduce the risk of this I would avoid sedative medication, long-acting opioid drugs, steroids, as well as antiemetics such as cyclizine and prochlorperazine which have central anticholinergic

effects. If sedation was needed at any point, I would use propofol and ensure capnography monitoring.

Postoperatively I would ensure they had some IV fluids running at a rate of 100 ml/hr as patients who have suffered hip fractures are often hypovolaemic. I would ensure frequent monitoring of blood pressure and I would check the Hb postoperatively. I would ensure that they had regular and PRN analgesia prescribed, as well as antiemetics, laxatives and venous thrombo-prophylaxis.

Postoperatively on the ward they should be having early physiotherapy input to help facilitate early mobilisation as well as a review by an ortho-geriatrician.

If the patient was adamant that they did not want a spinal anaesthetic, how would you approach performing a general anaesthetic for him?

This question is testing your ability to adapt and perform an alternative anaesthetic. Remember, as always, a safe and accepted technique is all the examiners are after as long as you can justify your rationale.

The principles regarding avoiding intraoperative hypotension, giving tranexamic acid, and avoiding drugs which are more associated with delirium would remain, as would the communication with the surgical team regarding any bone cementing.

I would again be keen to avoid any intraoperative hypotension and would closely monitor the patient's BP with regular non-invasive BP and would consider invasive monitoring if BP was on the lower side. I again would have a slow continuous metaraminol infusion to avoid having to use metaraminol boluses preventing there being peaks and troughs in the BP and I would be aiming to keep the MAP >70 mmHg.

With regards to induction, I would likely use a gas induction with sevoflurane as this would provide better cardiac stability. I would use age-adjusted MAC concentrations to avoid over-anaesthesia, as well as consider using bispectral (BIS) monitoring to assess the depth of anaesthesia.

I would use a second generation supra-glottic airway in this patient, as he has no history of gastroesophageal reflux and has been fasted. I would be aiming to maintain spontaneous ventilation throughout as this will minimise any risk of barotrauma, atelectasis as well as positive pressure ventilation-associated hypotension.

Post induction I would perform a fascia iliaca nerve block if the patient had not already had one or at least 6 hours had elapsed since they had received it.

Postoperatively I would manage the patient the same as previously: ensuring they had adequate analgesics, antiemetics and laxatives prescribed; had IV fluids running at a rate of 100 ml/hr; regular BP monitoring; Hb monitoring and early review by physiotherapy and an ortho-geriatrician.

In the past 10 years mortality from hip fractures has improved why do you think this is?

This question is testing your knowledge of the best practice guidelines for managing hip fractures.

Hip fractures are the commonest cause of death following accidental injury and as such there has been an increased focus on developing best practice guidelines. The reduction of mortality is likely multifactorial, but it is likely to have been aided by the

introduction of these guidelines and 'Best Practice Tariffs' which if achieved result in financial incentives for hospital trusts. There is an annual audit conducted by the National Hip Fracture database which focusses on six key performance indicators which include:

- Time to surgery withing 36 hours of presentation
- Assessment by a geriatrician within 72 hours of admission
- Being offered the NICE guideline specified type of surgery based on their type of fracture
- Assessment by physiotherapist on the day of or one day post-surgery, to facilitate prompt mobilisation
- Avoidance of delirium
- Return to patient's usual residence.

All the current guidelines focus on ensuring surgical fixation is offered promptly in patients who have suffered hip fractures, what would be appropriate reasons to delay surgery?

As you say early surgical fixation is preferable due to it being associated with improved mortality, better pain management and reduced rates of delirium; however, it is not always achievable. Reasons to delay a surgery would be:

- Significant anaemia with a Hb of less than < 80 g/L ; if this were the case I would like to give a preoperative transfusion aiming for a transfusion target of 90 g/L
- Severe hypo or hypernatraemia with sodium concentrations of <120 mmol/L or >150 mmol/L
- Severe hypo or hyperkalaemia with potassium concentrations of <2.8 mmol/L or >6.0 mmol/L
- Reversible coagulopathy
- Uncontrolled or sudden-onset left ventricular cardiac failure
- Cardiac arrhythmia with a rate of >120
- Uncontrolled diabetes
- Chest infection with signs of sepsis.

It would be advisable to perform surgery as soon as possible after the correction of the above issues.

What do you know about bone cement implantation syndrome?

Bone cement implantation syndrome (BCIS) can be defined as a hypoxia, hypotension or loss of consciousness occurring during cemented bone surgery. It most commonly occurs during bone cementation, insertion of a prosthesis, joint reduction or during relaxation of a limb tourniquet.

It can be categorised into being three grades depending on the severity of the hypoxia and hypotension, with grade 1 encompassing mild hypoxia and/or hypotension and grade 3 being a cardiac arrest.

The aetiology of BCIS is not fully understood but emboli from the bone in which cement is being inserted seems to be implicated as well as there being an immune-

mediated reaction to the cement, causing an increase in histamine and activation of the complement pathway.

Hypoxia is thought to be related to pulmonary emboli, and a proposed theory for the hypotension is that there is increased pulmonary vascular resistance; this in turn causes right ventricular dilatation which displaces the interventricular septum reducing volume of the left ventricle resulting in reduced cardiac output and hypotension.

Risk factors for the development of BCIS include increased age, poor physiological reserve, impaired cardiopulmonary function, pre-existing pulmonary hypertension, pathological and intertrochanteric fractures.

Reducing the risk of BCIS can be categorised into surgical and anaesthetic considerations.

From a surgical perspective this focusses on ensuring that the bone has been carefully washed using a pressurised lavage system to remove any bone marrow and endosteal fat, which will then be suctioned by a suction catheter. Cementing should be performed using a cement gun and excess manual pressurisation should be avoided.

From an anaesthetic perspective you should ensure that the patient is well hydrated, and there should be close communication with the surgical team when cementing is due to occur so that you can be vigilant for BCIS.

If BCIS is suspected it should be managed as an emergency, the operating team should be informed and implantation should be stopped, help should be called, the patient should be assessed with an ABCDE approach. The patient should have their O₂ concentration increased to 100% and hypotension should be managed with vasopressors as well as fluid boluses to ensure adequate preload.

Further Reading

Donalton AJ, Thomson HE, Harper NJ, Kenny NW. (2009). Bone cement implantation syndrome. *British Journal of Anaesthesia*, 102, 12–22.

Royal College of Physicians Facing new challenges — the NHFD report on 2020 (January–December 2020). London: RCP, 2021.

Scottish Hip Fracture Anaesthetic Recommendations v1.0, 2020 M.R. Checketts, Consultant Anaesthetist, Ninewells Hospital and Pavan Bangalore, Consultant Anaesthetist, Ninewells

Hospital, Incorporating feedback from anaesthetic representatives from all, NHS Scotland health boards. Consultation period Oct–Dec 2020.

Shelton C, White S. (2020). Anaesthesia for hip fracture repair. *BJA Education*, 20(5), 142.

White SM, Moppett IK, Griffiths R, Johansen A, Wakeman R, Boulton C, Grocott, MPW. (2016). Secondary analysis of outcomes after 11,085 hip fracture operations from the prospective UK Anaesthesia Sprint Audit of Practice (ASAP-2). *Anaesthesia*, 71 (5), 506–514.

2.9.1 Principles of Neonatal Physiology – Helen L Jewitt

This subject would lend itself well to a clinical science physiology viva or could be approached via a specific case scenario in the clinical viva. Don't be put off if you have little or no experience of anaesthetising neonates, a good understanding of the main principles and their relevance to anaesthetic practice will allow a confident answer.

How does neonatal physiology differ from that of the adult?

Begin by discussing normal physiology in the term neonate. An opening statement is very useful to show that you understand the question and have a clear idea of how your answer will be subdivided. This question is best approached with a systems classification, ensuring the important physiological differences between neonates and older patients are presented in an organised way.

A neonate is defined as a child within 28 days of birth. The child has undergone several physiological changes to adapt to life outside of the uterus. Despite these adaptations, there are many important physiological differences between neonates and older patients which have a significant impact on anaesthetic practice.

Start with the cardiovascular system as some of the most important factors of relevance to anaesthetic practice are found here.

Neonates have a higher heart rate and lower blood pressure in comparison to adults. An average heart rate of 120–180 bpm and average systolic blood pressure of 50–90 mmHg would be expected in a healthy neonate. The neonatal cardiovascular system is subject to increased demand and has a limited ability to respond to change as compared to an adult. The increased demand is a consequence of the neonate's need to maintain a stable body temperature via production of heat energy. This results in an oxygen consumption of 7 ml/kg/min which is twice that of an adult. Cardiovascular compensation is limited by the neonate's relatively fixed stroke volume. An increase in cardiac output is brought about largely by an increase in heart rate. Parasympathetic tone predominates and bradycardia is common, most importantly as a reflex response to hypoxia as well as vagal stimulation such as tracheal suction.

What is the circulating volume in a neonate and what is the significance of this?

A circulating volume at birth of 90 ml/kg corresponds to a total blood volume of 300–400 ml in an average sized neonate. Therefore, intraoperative bleeding of apparently small volumes can rapidly lead to loss of a significant proportion of total volume. Given

the relatively fixed cardiac output, this presents with tachycardia but may rapidly progress to cardiovascular collapse.

What is meant by the term 'transitional circulation'?

This question tests your knowledge of the changes which take place around the time of delivery to convert the fetal circulation into neonatal circulation.

At birth significant cardiovascular changes take place. These occur predominantly due to the rapid changes in both systemic and pulmonary vascular resistance. These are in turn brought about by two main events; the clamping of the umbilical cord and the generation of negative intrathoracic pressure from the first gasps.

During fetal life, systemic vascular resistance is low due to the presence of the low resistance feto-placental unit. Following clamping of the umbilical cord SVR increases markedly. In contrast pulmonary vascular resistance is very high in the fetus and falls as the first breaths are taken. This results in increased blood flow through the pulmonary circulation and back to the left atrium. This causes pressure in the left atrium to exceed that in the right atrium. The establishment of a pressure gradient provokes functional closure of the foramen ovale. The second adaptation to a continuous pulmonary-systemic circulation is the closure of the ductus arteriosus. This closes under the influence of the increased partial pressure of oxygen in the blood in the pulmonary artery. Closure of both ducts is initially functional only and is potentially reversible.

It is possible for a normal neonate to revert to a fetal pattern circulation in certain pathological circumstances. Hypoxia, hypercarbia and acidosis can cause constriction of the pulmonary vasculature, raising pulmonary vascular resistance. This can result in opening of the foramen ovale and ductus arteriosus with right-to-left shunting. This can lead to a self-perpetuating cycle of worsening hypoxia and worsening shunt. This is known as a transitional circulation or persistent fetal circulation.

What are the important factors to be considered in the respiratory system?

In the neonatal respiratory system there is incomplete development of the alveoli, producing relatively stiff lungs and a compliant chest wall. Work of breathing is higher and central responses to hypercarbia and hypoxia are immature compared to adults.

The functional residual capacity is small because the compliant chest wall is pulled inwards by the elastic recoil of the lungs. Tidal volume lies close to or within the closing range and airway collapse readily occurs producing intrapulmonary shunt and hypoxaemia. This can be reduced with the application of continuous positive airway pressure (CPAP). The horizontal alignment of the ribs in the neonate means they are unable to generate the 'bucket handle' movement seen in inspiration in adults. Breathing is predominantly diaphragmatic and the diaphragm is prone to fatigue given the reduced number of type 1 'fatigue-resistant' muscle fibres. Raised abdominal pressures and in particular a distended stomach can splint the diaphragm and precipitate respiratory failure. In a comparable situation to that in the cardiovascular system the resting respiratory rate is high, the capacity for an increase in tidal volume is minimal and compensation can only be achieved by an increase in respiratory rate.

The neonate is born with predominantly fetal haemoglobin, approximately 70% of all haemoglobin. This form of haemoglobin favours oxygen delivery to hypoxic tissues and

is a leftward shift in the oxyhaemoglobin dissociation curve. The proportion of adult haemoglobin (HBA2) increases during the neonatal period.

How does the central nervous system of a neonate differ?

In the central nervous system development is incomplete at birth. Myelination begins prior to full gestation and continues throughout the first year of life.

Normal intracranial pressure is lower than that of adults at 2–4 mmHg. The skull is less rigid than that of an adult due the presence of unfused sutures and fontanelles. An increase in intracranial pressure can be compensated for to an extent by expansion of sutures and bulging of fontanelles before cerebral insult occurs.

Cerebral blood flow in neonates is higher than that in adults with autoregulation occurring at lower mean arterial pressure, approximately 30 mmHg. In premature neonates autoregulation is absent and cerebral perfusion is pressure dependent, in addition the cerebral blood vessels are thin walled and this increases the risk of cerebral haemorrhage.

The blood–brain barrier is underdeveloped and more permeable leading to increased sensitivity to endogenous products such as bilirubin resulting in kernicterus. Iatrogenic agents may have a varied though often more potent effect as partially ionised molecules such as opiates, barbiturates and benzodiazepines can freely pass the barrier.

It was previously thought that the relative immaturity of the nervous system meant that neonates did not experience the sensation of pain. This has now been demonstrated to be incorrect with characteristic behavioural and physiological responses recognised in neonatal patients experiencing pain. These include tachycardia, hypertension, grimacing, crying and restlessness and can be scored using age specific pain scoring tools.

The conus medullaris terminates at the level of L1/L2 in neonates. Neuroaxial anaesthesia reduces the risk of postoperative apnoea in neonates and can be safely performed; however, their duration of action can be more unpredictable in comparison to adults. Spinal CSF volumes are approximately 2 ml/kg^{-1} at term, this was previously thought to be much higher; however, this has been disproved with the use of MRI

How is temperature regulation achieved in the neonate?

Neonates have very different mechanisms for maintenance of a stable body temperature. They lose heat readily due to a high surface area to weight ratio and are unable to shiver. Heat is produced by the metabolism of brown fat in a process known as non-shivering thermogenesis. Brown fat constitutes 5–6% of the body weight of a neonate and is located around the scapulae, kidneys and mediastinum. The metabolism of brown fat is sympathetically mediated and is a significant contributor to the high oxygen demand placed on the neonatal cardiorespiratory system. Thermogenesis by this mechanism is ablated by beta blockade.

Can you think of any issues of relevance to the renal system?

In common with the lungs and central nervous system, the kidneys continue to develop after birth. In the neonatal period there is a reduced glomerular filtration rate, reduced capacity to concentrate urine and reduced tubular function compared to adults. This

results in a limited ability to either conserve water in the event of dehydration or to cope with excessive solute or solvent administration. Fluid balance must be carefully calculated based on weight, insensible and observed fluid losses and maintenance requirements.

The afferent arteriole in a neonate is sensitive to prostaglandins; therefore NSAIDs are avoided as they produce vasoconstriction and decreased renal blood flow and could cause renal impairment.

Are you aware of any adaptations required to drug doses in neonatal patients?

Both the pharmacodynamics and pharmacokinetics of many drugs are different in the neonate compared to the adult due to many of the physiological factors already mentioned. Total body water is proportionally increased in neonates compared to adults. Water-soluble drugs have a greater volume of distribution and higher doses may be needed to achieve the same clinical effect. An example of this is suxamethonium which has a dose of 2 mg/kg in neonates compared to 1 mg/kg in adults. Suxamethonium can influence the muscarinic acetylcholine receptors at the sinoatrial node producing profound bradycardia and even asystole. This can be opposed by giving intravenous atropine before the suxamethonium.

Enzyme pathways in the liver are not fully developed and this combined with renal immaturity can lead to delayed metabolism and excretion of drugs. This is reflected in a considerable prolongation of the half-life of morphine due to a reduced ability to produce glucuronide conjugates.

Circulating levels of plasma proteins such as albumin and alpha acid glycoprotein are reduced leading to a higher free fraction of some drugs such as local anaesthetics.

Further Reading

Ahmad N, Greenaway S. Anaesthesia for inguinal hernia repair in the newborn and ex-premature infant. *BJA Education*. 2018 18(7) 211–217.

Gormley S, Crean P. Basic principles of anaesthesia for neonates and infants. *British Journal of Anaesthesia Continuing Education in Anaesthesia, Critical Care and Pain*. 2001 1(5) 130–133.

Macrae J, Ng E, Whyte H. Anaesthesia for premature infants. *BJA Education*. 2021 21(9) 355–363.

Martin L. The basic principles of anaesthesia for the neonate. *Colombian Journal of Anesthesiology*. 2017 45(1) 54–61.

Rochette A et al. Cerebrospinal fluid volume in neonates undergoing spinal anaesthesia: A descriptive magnetic resonance imaging study. *British Journal of Anaesthesia*. 2016 117(2) 214–219.

2.9.2 Neonatal Resuscitation and the Effects of Prematurity – Helen L Jewitt

How do you identify a newborn baby requiring resuscitation?

A newborn baby that is blue, pale or floppy with a heart rate of less than 100 bpm and poor or absent respiratory effort requires resuscitation.

Can you think of some factors that make a neonate more likely to require resuscitation following delivery?

In some situations, the potential need for neonatal resuscitation can be anticipated. These include preterm births, delivery following fetal distress, meconium stained liquor and complications at the time of delivery such as shoulder dystocia or cord compression. Administration of general anaesthesia or opioid analgesia to the mother is also a risk factor. Rarely there may be an antenatal diagnosis of a congenital abnormality; however, increasingly these are recognised prior to delivery and a planned resuscitation can take place upon delivery.

What are the steps in resuscitation of a neonate?

Neonatal resuscitation follows an airway, breathing, circulation approach in the same way as resuscitation of older children and adults. There are some important differences in recognition of the unique physiological circumstances in the period immediately following birth. Although it is something that many anaesthetists will not have carried out in practice, a sound understanding of the Neonatal Resuscitation Guidelines will be expected.

The first step is to dry the baby and wrap it in warm dry towels. At the same time a rapid assessment of the baby can be made looking at colour, tone, respiratory effort and heart rate. If the child is blue or has poor tone, inadequate respiratory effort or a slow heart rate, the next step is to open the airway. The child's head should remain in a neutral position with a jaw thrust to optimise the patency of the airway. The neck should not be extended as this can obstruct the airway.

Five inflation breaths should then be supplied using an appropriate sized facemask. Resuscitation can be commenced with air but oxygen should be immediately available in the event that the infant does not rapidly respond. The guidelines state that each breath should be maintained for 2–3 seconds at a pressure of 30 cmH₂O. Where possible, PEEP should be maintained at around 5 cmH₂O during the rescue breaths. If the infant is being resuscitated on a resuscitaire there is a pressure gauge to measure the pressure applied with each inflation breath. In practice ensure that sufficient pressure is applied to inflate the infant's chest.

If there is not adequate aeration of the lungs during the inflation breaths then repositioning the neonate's head and a two person approach to mask ventilation can be trialled. Suctioning of the airway should only be carried out under direct vision. An appropriately sized guedel airway or LMA can be used if needed when difficulty is encountered.

After this step, reassessment of colour, tone, respiratory effort and heart rate should take place. If the finding is of a heart rate of greater than 60 breaths/minute but absent respiratory effort, mask ventilation at a rate of 30–40 breaths/minute should continue until the baby is breathing effectively.

If there is chest expansion with the inflation breaths but the heart rate fails to recover to more than 60 beats/minute then chest compressions should be commenced. These are best delivered with two hands encircling the neonate's chest allowing compressions to be delivered with two thumbs. The ratio of compressions to breaths is 3:1.

If the neonate remains unresponsive despite the above steps consideration should be given to drugs and intubation. Vascular access can be secured via the umbilical vein, an intraosseous cannula or peripheral intravenous access (although this may be difficult).

If the heart rate does not recover to more than 60 beats/minute despite an open airway, effective ventilation and chest compressions for 30 seconds and then adrenaline should be considered.

During this type of scenario it is extremely rare that the cardiac rhythm is anything other than asystole resulting from hypoxia. An exception to this is in infants with congenital heart disease. In this event shockable rhythms may rarely be encountered. In this event 4.5 cm diameter pads should be applied to the infant in anterior and posterior positions. An energy of 4 J/kg is appropriate.

What would be an appropriate size endotracheal tube in a term neonate?

For an average sized neonate an endotracheal tube with an internal diameter of 3.5 mm would be appropriate.

Describe the aspects of neonatal physiology of greatest relevance to anaesthesia.

Use a systems approach to summarise these points.

The airway of a neonate requires careful management due to a large head, prominent occiput, large floppy epiglottis and narrow calibre conducting airways. The functional residual capacity is small and is exceeded by closing capacity. Neonates have a high oxygen demand and this in combination with a limited reservoir in the FRC means that rapid desaturation takes place. Neonates are unable to significantly increase their tidal volume and an increase in minute volume is produced by increasing respiratory rate. Inspiration is predominantly driven by the diaphragm, with weak intercostals and accessory muscles. Abdominal distension due to bag and mask ventilation or intra-abdominal pathology can severely impair respiration.

The cardiovascular system of a neonate has limited capability to increase cardiac output as stroke volume is relatively fixed. Compensation is achieved by an increase in heart rate.

The kidneys of a neonate are not fully developed, and they show impaired concentrating ability and tubular function. For this reason both dehydration and excessive fluid loads are poorly tolerated.

Hepatic enzymatic pathways are not fully active at birth; therefore the metabolism of some drugs may be prolonged.

Neonates are very susceptible to the effects of sedative drugs such as benzodiazepines, barbiturates and opioids. This is due to an immature blood-brain barrier and delayed hepatic inactivation of the drug.

A 10-week-old infant presents for inguinal herniotomy. He was delivered prematurely at 32 weeks gestation. What are the anaesthetic implications of prematurity?

It is not expected that all candidates will have direct experience of anaesthetising preterm infants but the examiner will look for a good theoretical understanding of neonatal anaesthesia and the specific problems of anaesthetising premature infants.

An opening statement shows that you have organised your thoughts.

This case raises the general problems common to anaesthetising neonates in addition to the issues specific to preterm infants. The anaesthetic history should focus on a full history from the parents of the infant and review of the medical notes. Particularly important information includes current post-conceptual age and complications since delivery such as the need for intubation, CPAP or supplemental oxygen.

Now move on to the problems specific to anaesthetising a preterm infant.

In addition to respiratory considerations for any neonate, it is especially important to maintain adequate ventilation in previous preterm infants. Depending on the ventilatory support, if any, they needed post-delivery, ex-preterm infants may have higher airways resistance. In addition, a previously instrumented airway should always raise the suspicion of tracheal stenosis requiring a smaller than anticipated tracheal tube.

Postoperative apnoea is common in infants of less than 60 weeks post-conceptual age. This infant would fall into this category and should be monitored postoperatively in a neonatal unit or high dependency area where continuous apnoea monitoring can be used. The anaesthetic technique could include the use of neuraxial anaesthesia to augment analgesia in order to reduce the risk of postoperative apnoea from opiate use.

Preterm infants have thin skin and limited subcutaneous fat stores and are very vulnerable to losing heat. They should be minimally exposed for the duration of the procedure, with the use of active warming methods such as overhead radiant heaters.

Hepatic immaturity in preterm infants means that glycogen stores are limited and hypoglycaemia can readily occur. Preoperative fasting times should be minimised and intravenous maintenance fluids should contain dextrose as well as electrolytes.

What do you know about retinopathy of prematurity?

How can the risk be minimised?

Retinopathy of prematurity is due to abnormal development of blood vessels in the neonatal retina. These vessels can invade the vitreous humour and are prone to haemorrhage causing scarring and detachment of the retina. Prematurity is the greatest risk factor for this problem, but it is exacerbated by the administration of high levels of oxygen. For this reason oxygen saturations should be maintained with the lowest accepted FiO_2 to maintain stability intraoperatively.

Further Reading

Fawke J Resuscitation Council UK Newborn Resuscitation and Support of Transition of Infants at Birth 2021.

Kariuki E, Sutton C, Leone T Neonatal resuscitation: Current evidence and

guidelines. *BJA Education*. 2021 21(12) 479–485.

Macrae J, Ng E, Whyte H Anaesthesia for Premature Infants *BJA Education*. 2021 21 (9) 355–363.

2.9.3 Anaesthesia in Infancy and Childhood – Jade A Loughran and Sarah F Bell

The developmental changes that occur during infancy and childhood have significant implications for the anaesthetist. They are regularly examined in the viva. A good understanding of the anatomical and physiological changes is vital.

You are involved in the preoperative assessment and anaesthetic planning for a premature neonate born at 28 weeks gestation who requires a laparotomy for necrotising enterocolitis. The baby is currently intubated on intensive care and fluid resuscitation and electrolyte management are ongoing.

Let's start by classifying the different ages of paediatric patients. Can you tell me how we decide when a neonate has become an infant or child?

A neonate is a baby within 44 weeks from the date of conception. This description therefore includes premature babies and full term babies up to 28 days old. An infant is described as being over 44 weeks post-conceptual age and up to 12 months. A child is aged 1 to 12 years.

Can you talk me through the anatomical differences between a paediatric and adult patient that occur in the respiratory system and why these are important to the anaesthetist?

It might help to remember everything by starting at the head and then working your way down the respiratory system.

Infants have a large head with a prominent occiput. The neck is short and the mandible relatively small. The tongue and epiglottis are large, which can predispose to airway obstruction. The optimal airway position in a neonate and infant is a neutral head position, differing from that of older children and adults. The larynx lies at the level of the 3rd or 4th cervical vertebrae in infants. This is high when compared to the adult level of the 5th or 6th cervical vertebrae. The higher position of the larynx and shape of the epiglottis mean that it may be easier to intubate an infant with a straight laryngoscope blade rather than a curved blade. The narrowest part of the paediatric airway is the cricoid cartilage, rather than the cords as in the adult. It is this anatomical difference that means uncuffed endotracheal tubes are widely used in paediatric anaesthesia. The size of the tracheal tube for a neonate is approximately 3–3.5 mm, although this may vary. A small leak should always be present to ensure minimal risk of trauma to the tracheal lining. The formula for guiding tracheal tube size is age divided by four, plus four.

The trachea may be less than 4 cm in length in the neonate. This can lead to endobronchial intubation if the anaesthetist is not meticulous when positioning and securing the endotracheal tube. The formula for the length of an oral tracheal tube is age divided by 2 plus 12. This gives the length at the lips. For nasal tubes the number to add is 15 rather than 12.

The carina branches at the same angle on both sides of the chest in infants. The airways are also much smaller in diameter than in adults. Any additional narrowing will lead to a marked increase in resistance to air flow. In infants, the intercostal muscles and diaphragm are weaker, with fewer type one fibres. They therefore fatigue faster than adults. The diaphragm is the main muscle responsible for respiration and so anything that compromises its efficiency (such as abdominal distension) will cause breathing difficulties. The chest wall is also more compliant and so recession is a marker of respiratory effort.

What about the physiological changes that occur in the respiratory system?

Paediatric patients have higher oxygen consumption than adults. A neonate has an oxygen consumption of 6–9 ml/kg/min when compared to the adult 2–3 ml/kg/min. The alveolar minute volume is increased in order to meet this increased demand. Since the tidal volume of approximately 7 ml/kg is similar for paediatric and adult patients the increase is achieved by changes in respiratory rate. A neonate's respiratory rate ranges from 40 to 60 breaths/minute, a child's 20–30 and the adult range is 12–24 breaths/minute.

The significant difference in oxygen consumption means that neonates will desaturate faster than adults.

Can you talk me through the changes in functional residual capacity and closing capacity?

The functional residual capacity is similar for paediatric and adult patients at about 30 ml/kg. In infants, neonates and young children closing capacity encroaches on the functional residual capacity, leading to airway closure at the end of expiration. In order to counteract this, partial adduction of the cords occurs during expiration, producing physiological CPAP. When anaesthetising an infant or young child using a spontaneously breathing technique, CPAP will aid oxygenation and reduce the work of breathing. When a paediatric patient is fully ventilated, the respiratory rate and tidal volume should be set appropriately. PEEP can be used to avoid airway closure and improve oxygenation.

What can you tell me about the control of respiration in a neonate or young infant?

The neonatal respiratory control is immature. The peripheral chemoreceptor response to hypoxia and the central chemoreceptor response to carbon dioxide are both weak and blunted. Apnoeic episodes may occur up to 60 weeks post conception. These patients need to be monitored postoperatively with an apnoea monitor.

Now, let's move on to the cardiovascular system. Can you briefly summarise the changes that occur at birth?

This topic is also discussed in the neonatal physiology podcast. Try and talk through the changes as they occur so that the examiner knows that you have a strong grasp of the basics.

At birth the umbilical cord clamping and the first breath switch the site of gas transfer from the placenta to the lungs. As the first breath is taken the lungs expand and the pulmonary vascular resistance falls by 80%, increasing pulmonary blood flow dramatically. The systemic vascular resistance increases due to the exclusion of the large, low-resistance placental vascular bed when the umbilical cord is clamped. The changes in pulmonary and systemic vascular resistance alters the pressure gradient between the right and left sides of the heart, causing the foramen ovale to close and blood flow through the ductus arteriosus to be reversed. The increase in the partial pressure of oxygen and reduction in prostaglandin E2 stimulates closure of the ductus arteriosus.

Over what time period do the foramen ovale and ductus arteriosus close?

The foramen ovale closes as soon as the pressures reverse within the heart, but it can reopen within the next five years of life. The ductus arteriosus contracts in the first few days of life and then fibroses within a month.

Can you tell me about the anatomical and physiological differences between the paediatric and adult cardiovascular system?

The neonatal heart has non-compliant, stiff ventricles. At birth the right and left ventricles are similar in size. The cardiac output is high to meet the high oxygen consumption. In a neonate the cardiac output is 200–250 ml/kg/minute compared to 80 ml/kg/minute in an adult. Stroke volume is relatively fixed and so the only way to increase the cardiac output is by increasing the heart rate. The average heart rate in the neonate is 120 beats/minute, falling to 100 in childhood and 75 as an adult.

The blood pressure is lower in the paediatric population. A neonate would have a systolic pressure of 50 to 90 mmHg, a child 95 to 110 mmHg and an adult 95 to 110 mmHg. Because of the dependence on heart rate to maintain cardiac output, bradycardias are poorly tolerated in paediatric patients.

What about the differences in the haematological system?

The blood volume is greater in a neonate than an adult at approximately 90 ml/kg. This falls to 80 ml/kg in a child and then 70 ml/kg in an adult.

Extracellular fluid volume is also greater in the neonate as 40% of body water is extracellular in the neonate compared to about 20% in the adult. By the age of two this difference has disappeared. The greater metabolic rate results in a faster turnover of extracellular fluid in the neonate and infant. Interruptions in normal intake can lead to the rapid onset of dehydration.

How would you approach fluid management in a child?

I would consider fluids in terms of maintenance, replacement and deficit. With regards to maintenance fluids I would use the 4, 2, 1 rule. This means for the first 10 kg weight the child will require 4 ml/kg/hr, for the next 10 kg 2 ml/kg/hr and for every subsequent 10 kg 1 ml/kg/hr. Replacement fluid includes all ongoing losses. These losses should be closely monitored, for example weighing swabs and measuring suction fluid. For abdominal surgery approximately 10 ml/kg/hr is lost. Calculation of the fluid deficit requires clinical assessment of the child. A 5% loss might present as dry skin and mucous membranes, 10% loss would lead to cool peripheries, a depressed fontanelle and oliguria, and 15% loss would lead to changes in conscious level and hypotension. Replacement is calculated as 10 ml/kg multiplied by the percentage deficit. This should be given over 24 hours.

What fluids should be used if a child needs fluid resuscitation?

Intravenous fluid resuscitation should be with crystalloid that does not contain glucose, with a sodium content of 131–154 mmol/L, either 0.9% sodium chloride or Hartmann's solution. A bolus of 20 ml/kg is often used, although this may need to be

reduced if the child has cardiac or renal disease. In cases of trauma, 10 ml/kg fluid boluses are used.

Can you tell me anything about haemoglobin in neonates?

At birth the neonate has fetal haemoglobin. The fetal oxyhaemoglobin curve is shifted to the left due to a reduction in 2, 3-DPG. It is therefore better suited to take up oxygen at the lower partial pressures present in the placenta (but oxygen is also less readily released). In order to maintain oxygenation to the tissues the neonate has a high haemoglobin concentration of about 170 g/L, the blood volume is increased and the cardiac output is high.

A physiological anaemia occurs at about 3 months when fetal haemoglobin is replaced by adult HbA.

When might you consider transfusing a bleeding infant?

Generally I would give blood if the haematocrit fell to less than 25%, or the estimated blood loss was greater than 20% of the blood volume.

What about changes that occur to clotting factors and platelets?

The vitamin K dependent clotting factors 2, 7, 9 and 10 are deficient in the first few months. Therefore vitamin K is given to newborn babies. Platelet function is also reduced.

Why are neonates and infants at risk of hypothermia?

The paediatric patient is at risk of hypothermia due to increased heat loss and poor compensatory mechanisms. Heat is lost by thermal conduction from thin skin and lower levels of body fat. A high body surface area to weight ratio and increased minute ventilation further contribute to the loss. Infants less than three months old undergo non-shivering thermogenesis. Production of heat from brown fat is inefficient and actually increases oxygen consumption. In older infants and children shivering is ineffective due to limited muscle mass. Vasoconstriction is also poor.

Why is this important to the anaesthetist?

This question could also lead onto the effects of hypothermia. This is covered in the hypothermia podcast and only a brief summary given in the following answer.

It is vital to pay careful attention to temperature and warming since hypothermia can cause large increases in oxygen consumption and detrimental effects on cardiac output, nervous and haematological systems. As anaesthetists we can avoid this by monitoring body temperature and considering: covering, avoiding exposure, increasing ambient temperatures, heating covers, warming fluids, HME's and circle breathing systems.

Can you tell me about any differences between the infant and adult central nervous system?

In neonates and infants the anterior fontanelle may be used to assess the intracranial pressure. This ossifies by about 18 months. The cerebral blood flow in a neonate is about 50 ml/100 g/min. It then rises significantly in childhood (to about 100 ml/100 g/min)

before returning to the lower levels during adulthood. Cerebral oxygen consumption is also much higher during childhood reaching values of 5.8 ml/100 g/min compared to adult levels of 3.5 ml/100 g/min.

The blood–brain barrier is incomplete at birth. The volume of CSF is proportionally larger in infants than adults (4 rather than 2 ml/kg). At term, the spinal cord terminates at L3. It then recedes to L1/2 by adolescence. The sacral hiatus is large and not ossified, making it an attractive location for caudal anaesthesia. Myelination is incomplete until 1–3 years. The sympathetic nervous system is not fully developed until 6-years and so bradycardias are relatively common.

How do the actions of general anaesthetic agents differ in the paediatric patient?

With regards to the inhalational agents, both induction of and emergence from anaesthesia are more rapid due to the greater alveolar ventilation. MAC is reduced in neonates, while in infants and children it increases by up to 30% of that of adults. In neonates the immature blood–brain barrier and reduced metabolism leads to increased sensitivity to barbiturates and opioids. Reduced doses are therefore required. In older children higher doses are needed.

Propofol infusions are not licensed for children below 3 years due to reports of neurological, cardiac, renal and hepatic impairment.

Do the muscle relaxants also have different effects?

Neonates and infants have increased sensitivity to muscle relaxants at the neuromuscular junction. A similar loading dose to adults needs to be given due to dilution of the drug by the larger volume of distribution. The reduced GFR, clearance and increased sensitivity leads to prolonged effects.

What can you tell me about the differences between the paediatric and adult renal system?

The glomerular filtration rate and renal blood flow are low at birth and gradually increase. The kidney initially has limited concentrating ability and mechanisms to maintain fluid, electrolyte and acid–base homeostasis. Values reach adult levels by about 12 months. Infants cannot handle large water or sodium loads. Urine output is normally 1–2 ml/kg/hr.

And what about the liver?

At birth the vitamin K-dependent clotting factors are low as are glucose storage levels. Physiological jaundice may occur in the neonate due to increased red blood cell breakdown with limited ability to metabolise unconjugated bilirubin. Phase 1 and 2 reactions take 2–3 months to reach full activity.

Can you give me some guidelines for fasting for the paediatric patient?

The current guidelines in my hospital are that no food should be consumed for 6 hours preceding the operation, including formula and cow's milk. No breast milk should be taken for 4 hours and no clear liquid for 2 hours.

Finally, what psychological changes might occur and how might they affect the anaesthetist?

In infants less than 6 months no separation anxiety occurs. From 6 months to 4 years behaviour is unpredictable and separation anxiety is seen. It is important to try and keep the carer with the patient as much as possible. School children are upset by the thought of the surgical procedure, its mutilating effects and pain. We need to bear this in mind when explaining the induction, operation and postoperative analgesia.

With regards to adolescents, they find the loss of control and thought of pain upsetting. Their reliance on a carer is varied.

It is important to use the preoperative visit as an opportunity to assess the child from this point of view as well as the formal anaesthetic assessment, and to take time to build up a rapport with both the patient and their carers.

Further Reading

NICE guideline NG29. Intravenous fluid therapy in children and young people in hospital. 2015 (updated 2020).

Sodhi P and Fiset P. Necrotising enterocolitis. *Continuing Education in Anaesthesia Critical Care and Pain*. 2012;12(1):1–4.

2.9.4 Pyloric Stenosis – Penelope S Colter, Sarah F Bell and Caroline SG Janes

This would generally form part of a clinical structured oral exam. The biochemical abnormalities are frequently asked, so make sure that you can explain them thoroughly.

What is congenital hypertrophic pyloric stenosis?

Pyloric stenosis is a congenital narrowing of the gastric outflow tract caused by hypertrophy of the circular smooth muscle of the pylorus. The cause is unknown but there is a genetic predisposition.

The condition occurs in 1.5 per 1,000 births. It is the most frequent cause of intestinal obstruction in infancy and the commonest small baby condition treated outside a specialist centre. Boys are affected much more than girls, in a ratio of 4 to 1. A total of 40–60% of cases occur in first-born children. The condition is also more common in the Caucasian population.

Pyloric stenosis can also be acquired in adults as a result of gastric carcinoma or chronic peptic ulceration. These conditions will not be discussed in this podcast.

How does pyloric stenosis present?

The patient usually presents at 4 to 6 weeks of age with worsening symptoms of persistent, projectile, non-bilious vomiting. The infant typically feeds well but then vomits after each feed. The infant will be hungry and may have lost or failed to gain weight.

On examination a hard ‘olive-like’ mass may be palpable. This is classically 1–2 cm in diameter and located in the right upper quadrant at the lateral edge of rectus abdominus muscle. The infant is often dehydrated.

What might arterial blood gas and urinalysis show?

Arterial blood gas will demonstrate a marked metabolic alkalosis with hypokalaemia and hypochloraemia.

Urinalysis will show acidic urine with high levels of potassium.

How can the blood gas results be explained?

This is complicated. Try and keep your explanation simple. By talking through the gastrointestinal changes followed by the renal compensation and then the respiratory alterations you will be able to remember all the key facts.

The blood gas results are due to a combination of gastrointestinal, renal and respiratory changes.

With regards to the gastrointestinal system, it is important to know the components of the gastric and small bowel secretions and the differences between normal vomiting and vomiting in pyloric stenosis. Gastric fluid is rich in hydrogen chloride. This is neutralised by the bicarbonate ions secreted by the small bowel. In normal vomiting there is mixing of gastric and small bowel fluid. There is therefore no change in the plasma pH but fluid and electrolyte loss will lead to dehydration. In pyloric stenosis the vomit does not contain bicarbonate due to pyloric obstruction preventing mixing. There is therefore only hydrogen and chloride ion loss.

Due to these biochemical changes, the kidney is presented with a large bicarbonate load.

This exceeds the absorptive threshold and so alkaline urine is initially seen.

Prolonged vomiting leads to hypovolaemia and dehydration. This causes activation of the renin–angiotensin–aldosterone axis in an attempt to restore circulating volume. The aldosterone acts on the kidney to retain sodium at the expense of potassium and hydrogen ions. This leads to the production of paradoxical acid urine and worsening hypokalaemia and metabolic alkalosis.

The infant may attempt to compensate for the metabolic alkalosis by using the respiratory system. They may hypoventilate to produce hypercapnia, but this will never be sufficient to correct the alkalosis as the hypoxic drive will be triggered.

What other biochemical or haematological changes might be seen?

Hypoglycaemia, haemoconcentration, mild uraemia and unconjugated hyperbilirubinaemia may be seen.

A 5-week-old infant is brought into your hospital and is diagnosed with pyloric stenosis. He is moderately dehydrated. Arterial blood gases demonstrate a pH 7.6, chloride of 80 mmol/L and potassium of 3.0 mmol/L. The bicarbonate ion concentration is 32 mmol/L. The child was born at term by normal vaginal delivery. There are no other cases on the emergency list and the surgeon is keen to proceed as soon as possible.

What are the issues with this case?

There are a number of issues with this case. These can be divided into problems specific to the pyloric stenosis and the general problems with anaesthetising a young infant.

With regards to the issues relating specifically to the condition these include: the markedly deranged acid–base status, the effects of dehydration and the increased risk of regurgitation due to the obstruction. Pyloric stenosis correction is not an emergency. The infant should be fully resuscitated and the electrolyte abnormalities and alkalosis corrected prior to surgical intervention.

The challenges of anaesthetising a small infant include the altered anatomy and physiology, the presence of anxious parents, difficulty obtaining intravenous access and the altered drug dosages.

This case should be undertaken with the support of a consultant anaesthetist experienced in paediatric anaesthesia.

How would you go about resuscitating this infant?

The infant needs to be resuscitated in an area where the nursing staff are trained in the management of these complex cases. This might be a paediatric surgical ward or high dependency unit. The parents should be with the infant whenever possible.

I would assess the degree of dehydration and obtain intravenous access. A full blood count, renal function tests, liver function tests and group and save should be sent, along with blood gases. These will aid the resuscitation and help to decide on the required potassium supplementation. Regular blood gases will help to ascertain the success of resuscitation.

A nasogastric tube should be placed.

Regular observations need to be taken. These include respiratory rate, oxygen saturations, heart rate, blood pressure, conscious level, urine output and ongoing gastric losses.

How would you assess the degree of dehydration in this infant?

Assessment can be divided into history and examination. In my history I would ask the parents how much the infant had been vomiting and how much he had been taking orally. I would ask about any diarrhoea and whether the infant had been febrile. I would also enquire about whether the parents had noticed a change in how often the infant was wetting his nappy and whether they had observed any change in the infant's alertness or conscious level.

I would examine the infant looking for dry mucous membranes, a sunken fontanelle and eyes, tachycardia, prolonged capillary refill time, decreased conscious level and hypotension. As a guide, the child with dehydration but no shock can be estimated to be 5% dehydrated. If shock is present then the child is at least 10% dehydrated.

How would you approach fluid management in this case?

Fluid management can be divided into resuscitation fluids, maintenance fluids and ongoing losses.

Resuscitation fluids should be calculated by assessing the degree of dehydration. The volume of fluid required to replace this can then be calculated by multiplying the percentage dehydration by the weight of the infant multiplied by ten. Hartmann's solution or 0.9% normal saline are both appropriate fluids. Half of the fluid deficit should be corrected within the first 24 hours followed by half over the second 24 hours.

In addition to this, maintenance fluids are given. This may also be Hartmann's solution or 0.45% saline with 5% dextrose. The four–two–one rule can be used to decide the amount of fluid to give per hour to a paediatric patient. Four millilitres per kilogram is given for the first 10 kg of body weight of the infant, followed by 2 ml/kg for the second 10 kg body weight and then 1 ml/kg for each following 10 kg.

What is this regimen based on?

This regimen is based on the amount of water required to give formula feed orally, on the assumption that the required calorie intake is 100 kcal per kg per day. The water supplied is in excess of actual maintenance requirements. The calculation relies on good renal function to excrete the excess water. Outside the neonatal period, hypoglycaemia is rare but water load can be a problem. It may be preferable to use 0.9% normal saline with added glucose of 5 or 10%, checking frequently for hyperglycaemia.

Do you know how much potassium you would expect to add to the maintenance fluids?

It is recommended that 3 mmol/kg/24 hour of potassium should be added to the maintenance fluids, as guided by regular biochemical analysis of blood.

When would you be happy to proceed with anaesthesia?

I would want the infant to be normovolaemic. If the infant had been catheterised I would aim for a urine output of 1–2 ml/kg/hr.

Biochemical parameters should be within the following range before proceeding to surgery: pH 7.3–7.45; chloride 95–112 mmol/L; base excess -4 to 2.5 mmol/L; potassium 3.5–5.5 mmol/L.

How long does a pyloromyotomy take and how is it performed?

This procedure tends to take about 30 minutes to an hour. It may be performed open or laparoscopically. Laparoscopic surgery on infants is generally only performed in specialist centres.

How would you anaesthetise this infant?

A senior, experienced, paediatric anaesthetist should be present.

Prior to induction I would aspirate the nasogastric tube, attempting to empty all four quadrants of the stomach by moving the infant from supine, to left lateral decubitus, to prone, to right lateral decubitus. Aspirate the NG tube in each position.

Both inhalational and rapid sequence inductions with and without cricoid pressure have been described. In practice, classic rapid sequence inductions are not always appropriate in this age group. The patient is likely to need gentle bag-mask ventilation to prevent hypoxia and subsequent bradycardia given their higher rate of oxygen consumption and small FRC. Cricoid pressure may make intubation difficult or impossible. An adequate dose of muscle relaxant should be given.

I would secure the airway with an appropriately sized, microcuffed endotracheal tube. A size 3.5 mm tube would probably be adequate for this infant, but I would have a

3 mm and 4 mm sizes available. I would then hand ventilate the infant and check my tube position before connecting to a paediatric circle system using pressure-controlled ventilation.

I would maintain anaesthesia with sevoflurane, oxygen and air. I would avoid nitrous oxide in this case as it causes expansion of bowel gas.

Paracetamol, local anaesthetic infiltration and opioids can be given for analgesia.

Paracetamol can be given intravenously and rectally. The doses for an infant under 10 kg are 10 mg/kg TDS up to 30 mg/kg/day intravenously; or 15 mg/kg QDS up to 75 mg/kg/day rectally. Note the intravenous dose is further reduced to 7.5 mg/kg TDS intravenously for premature neonates >32 weeks corrected gestational age. Oral paracetamol 15 mg/kg QDS may be commenced once oral fluids are tolerated.

The dose of local anaesthetic needs to be calculated depending on the size of the infant. If levobupivacaine is used, the maximum dose is 2 mg/kg. Local infiltration can be performed by the surgeons. Ultrasound-guided transversus abdominis plane (TAP) blocks can be used, or a rectus sheath catheter for open pyloromyotomy with an incision near the umbilicus.

Morphine can be given intravenously at a dose of 50–100 mcg/kg, but this is rarely required.

The nasogastric tube can be removed at the end of surgery and the infant should be extubated in the left lateral position.

What would be your postoperative instructions for this infant?

Due to the potential risk of postoperative apnoeas up to 60 weeks post-gestational age, the infant should be monitored with an apnoea alarm and oxygen saturation probe for at least 12 hours.

Re-establishing feeding varies between centres, but there is a trend towards early feeding on demand. Some centres use a 2 hour nil by mouth period postoperatively. Intravenous maintenance fluids should be continued until feeding is established. The first feed should be clear fluid.

I would prescribe regular paracetamol for analgesia.

How can you assess pain in paediatric patients?

Pain assessment can be difficult in children due to communication and behavioural challenges. Specific pain assessment tools have therefore been developed for paediatric patients.

Assessment should be performed regularly with the support of the parents. Since pain is a subjective experience, it is important to seek the child's self-report of pain whenever possible. Neonatal assessment uses a combination of physiological and behavioural markers including: facial expression, body and limb movements, cry, sleeplessness, cardiovascular and respiratory changes. Examples of tools available are the Neonatal and Infant Pain Scale (NIPS) and the Neonatal Pain Agitation and Sedation Score (N-PASS).

Infant assessment is also purely observational. Behavioural pain indicators include: facial expression, irritability, unusual posture, screaming, sobbing or whimpering, reluctance to move, increased clinginess, loss of appetite and disturbed sleep pattern. Examples of tools available are the Face, Legs, Activity, Cry and Consolability scale (FLACC) and the Objective Pain Score (OPS).

Pre-school children may be able to self-report pain when given appropriate tools; for example, 'Faces' is a face-based scale with happy to crying faces to represent the pain scale. School children can usually communicate pain severity and location. They understand numerical concepts and so may use numerical rating scales. Other tools include faces, colour-based scales and visual analogue scales.

Finally, adolescents can communicate pain severity, location and intensity and can use visual analogue scales or numerical rating scales.

2.9.5 Intussusception – Penelope S Colter, Sarah F Bell and Caroline SG Janes

What is intussusception?

Intussusception is caused by the small bowel telescoping, as if it were swallowing itself. This usually occurs at the ileocaecal junction. Intussusception can lead to venous congestion, bowel oedema and intestinal obstruction.

What age group tend to present with this condition?

Intussusception occurs predominantly in infants and young children. Fifty per cent of cases occur in infants and only 10% of cases occur in children older than five.

Is intussusception seen more in boys or girls?

The condition is seen more often in boys.

What are the causes of intussusception?

Ninety per cent of cases are idiopathic. Intussusception is the most common cause of intestinal obstruction in the first year of life. In children over one, intussusception may be the first presentation for a number of pathologies such as Meckel's diverticulum, intestinal polyp, lymphoma, haemolytic uraemic syndrome, Henoch-Schonlein purpura or Peutz-Jegher's syndrome.

Can intussusception recur?

Yes, the recurrence rate is 5%.

Now let's review a case. An 8-month-old boy has been admitted to your hospital. His mother says that he has been intermittently crying and pulling his legs up to his chest for the past 2 days. She found a redcurrant jelly-like stool in his last nappy. He has vomited occasionally, and she has noticed that his nappies have not been wet the last few times she changed them.

Your examination reveals a quiet, pale baby. His heart rate is 150 beats/minute and capillary refill time is 4 seconds. His abdomen appears distended and the infant cries when you examine him.

How might an infant with intussusception present?

The classical features are of paroxysmal abdominal pain and blood and mucus in the stool. This is sometimes described as having a 'red currant jelly' appearance. The infant

has episodes of inconsolable crying and drawing up its legs. Vomiting and dehydration may be evident, potentially leading to shock. Abdominal distension can cause respiratory compromise. Infection, bowel infarction, bleeding and perforation may all occur.

On examination, a sausage-shaped mass may be palpable in the right side of the abdomen. The abdomen may be distended with guarding or rigidity. The child may be profoundly shocked.

What would be your differential diagnosis for this history?

The infant is most likely to be suffering from a gastrointestinal condition. Intussusception with dehydration and possible sepsis would be my main diagnosis. I would also consider gastroenteritis.

How do you diagnose intussusception?

Diagnosis is made from the history and examination and then investigations. The investigation of choice is an abdominal ultrasound. An air enema can be diagnostic as well as curative in up to 70% of cases. This intervention is contraindicated if a patient is in shock, has peritonitis or has perforated.

An abdominal X-ray may aid the confirmation of bowel obstruction and possible perforation.

How would you treat intussusception if air enema has failed?

The patient will need either a laparotomy or laparoscopy. Any necrotic bowel found will need to be resected.

Would you advise air enema or surgical intervention in this case?

This infant has evidence of shock because he has tachycardia, increased capillary refill time, reduced urine output and conscious level. The infant should be resuscitated as a matter of urgency and an emergency laparotomy should then be performed.

How would you resuscitate this infant and prepare them for theatre?

Try and describe the resuscitation as you would actually do it. This will hopefully help you remember everything!

Resuscitation should either be performed in a high dependency unit or in the anaesthetic room or recovery area prior to theatre. I would obtain intravenous access and perform a capillary or venous gas. I would send bloods for full blood count, urea and electrolytes, liver function tests and cross-match. I would regularly monitor the vital signs of the infant including the respiratory rate, oxygen saturations, heart rate, blood pressure, capillary refill, urine output and conscious level.

Fluid resuscitation is vital. I would give fluid boluses of 20 ml/kg 0.9% normal saline or a balanced crystalloid solution to restore the circulating volume. After each bolus I would reassess the infant to see whether further fluid was required. My reassessment would consist of reviewing the infant's observations, particularly the capillary refill time, heart rate and conscious level. The blood pressure might be maintained due to tachycardia and coexisting pain.

In addition to resuscitation fluids, I would give maintenance fluids calculated from the weight of the patient. Hartmann's solution can be used to provide this. If the blood results revealed any severe abnormalities in the urea and electrolytes then I would start correcting these prior to theatre.

I would place an NG tube, aspirate the stomach contents and then place the tube on free drainage.

If the infant's respiration were inadequate due to either the diaphragmatic splinting from abdominal distension or the effects of severe shock, I would consider intubating and ventilating the infant. This could be done in theatre.

How would you induce anaesthesia in this infant?

I would anaesthetise this infant with the support of a consultant paediatric anaesthetist. A general anaesthetic with endotracheal intubation and ventilation is required.

Prior to induction I would ensure that the infant had been adequately resuscitated. I would suction the NG tube and preoxygenate the infant. I would obtain monitoring as per the AAGBI standards and have all my drugs for resuscitation and induction available.

The options for induction of general anaesthesia are a gas or intravenous induction. I would prefer a rapid sequence induction with cricoid pressure. In practice, classic rapid sequence inductions are not always appropriate in this age group. The patient is likely to need gentle bag-mask ventilation to prevent hypoxia and subsequent bradycardia given their higher rate of oxygen consumption and small FRC. Cricoid pressure may make intubation difficult or impossible. I would use ketamine to induce the infant and then give a muscle relaxant. This could be either suxamethonium or an adequate dose of non-depolarising muscle relaxant such as rocuronium 1.2 mg/kg. I would then intubate the infant with a straight blade and pass a microcuffed endotracheal tube. I would check the position of the tube by auscultating the chest, observing the capnography trace and checking the length at the lips. I would ensure that the cuff pressure was checked.

What about maintenance?

I would maintain anaesthesia using an inhalational agent. Personally, I would use sevoflurane. I would then hand ventilate the infant, before connecting to a paediatric circle system using pressure-controlled ventilation. I would avoid using nitrous oxide in this case.

What would you give for pain relief?

I would use a combination of paracetamol, local anaesthetic techniques and opioids for analgesia.

Paracetamol can be given intravenously and rectally. The doses for an infant under 10 kg are 10 mg/kg TDS up to 30 mg/kg/day intravenously; or 15 mg/kg QDS up to 75 mg/kg/day rectally. Note the intravenous dose is further reduced to 7.5 mg/kg TDS intravenously for premature neonates >32 weeks corrected gestational age. Oral paracetamol 15 mg/kg QDS may be commenced once oral fluids are tolerated.

Non-steroidal anti-inflammatories may be an option provided the infant is adequately hydrated with good renal perfusion and no evidence of sepsis. The dose of

oral ibuprofen is 5 mg/kg QDS for 3–12 month old infants, and 10 mg/kg TDS for those older than 1 year. Diclofenac may be given orally or rectally at a dose of 1 mg/kg TDS (max 50mg) if aged >6 months and 8–11 kg in weight.

Opioids can be used intra- and postoperatively. Morphine can be used in judicious boluses or by infusion. It is preferable to fentanyl due to its greater water solubility. It is less lipid-soluble and so less cumulative and more predictable. Due to the risk of post-operative respiratory depression the infant should be carefully monitored. Local anaesthetic techniques such as wound infiltration or abdominal plane blocks could be used. The maximum safe dose of levobupivacaine is 2 mg/kg. An epidural could be considered but would often be contraindicated due to sepsis and/or coagulopathy.

What would be your concerns during the operation?

My main concerns during the operation can be divided into specific concerns regarding the laparotomy and general considerations for paediatric patients. Specifically, I would be vigilant for any surgical complications or haemorrhage. I would aim to maintain good communication with the surgeon.

Generally, I would want to closely monitor the infant and avoid excessive heat loss, give adequate doses of analgesics and anaesthetic agents and give appropriate fluids, taking into account the fluid losses during surgery.

I would aim to maintain the oxygen saturation, end-tidal carbon dioxide, heart rate, blood pressure, temperature and urine output all within normal levels throughout the operation. I would also check the blood sugar levels of the infant regularly to avoid hypoglycaemia.

The infant is at risk of hypothermia during the operation due to heat loss from the skin (aided by peripheral vasodilatation) and the surgical site. I would aim to increase the ambient temperature of the operating theatre and anaesthetic room prior to and during surgery. I would use a heated mattress, Bair Hugger and cover the infant's head in order to avoid further heat loss from the skin. Bubble wrap and plastic drapes can also be used. I would warm the fluids given to the infant and use a HME to reduce the heat loss from the respiratory system. Use of a circle system also provides warm, fully saturated gas and is therefore superior to a T-piece system.

How would you approach intraoperative fluid management?

I would try and consider fluids in terms of pre-existing deficits, maintenance requirements and ongoing losses.

I would resuscitate the infant prior to induction of anaesthesia, thus addressing the pre-existing deficit.

With regards to maintenance fluids I would calculate this from the weight of the infant: 4 ml/kg is given for the first 10 kg of body weight of the infant, followed by 2 ml/kg for the second 10 kg of body weight and then 1 ml/kg for each following 10 kg. I would use Hartmann's solution as a continuous infusion, although this may need to be changed depending on the blood results. A glucose solution may also be required depending on the blood sugar measurements.

The ongoing losses would be mainly due to the operation. Blood loss should be closely monitored. As a general guide, approximately 10 ml/kg/hr fluid will be required to compensate for evaporation during a laparotomy. This can be replaced using

Hartmanns solution. If the infant is septic, boluses totalling 40 mg/kg are not unusual. If significant blood loss occurs, then I would give blood and consider the need for clotting factors or FFP.

What would be your postoperative plan for this patient?

If the infant requires ongoing ventilation due to respiratory compromise, septic shock, blood loss or surgical complications then they will need to be nursed in a paediatric intensive care unit. Otherwise, I would want the patient to be nursed on a high dependency unit, in order to closely monitor the vital signs and pain levels of the infant. Respiratory depression may occur due to the effects of opioid analgesics.

Regular blood tests will be necessary to check the full blood count and urea and electrolytes. Intravenous fluids should be continued until the infant is able to feed. Blood transfusion may also be required.

2.9.6 Oesophageal Atresia, Diaphragmatic Hernia and Exomphalos – Helen L Jewitt

These are paediatric surgical problems, which many Final FRCA candidates may not have encountered in their training. You are unlikely to encounter questions on more than one of these problems within a viva. A structured understanding of each problem and the main anaesthetic considerations relating to it will ensure you tackle a potentially difficult question well.

You are on call for paediatric surgical emergencies and are asked to assess a term neonate with oesophageal atresia. What do you understand about this condition?

Oesophageal atresia (OA) and tracheo-oesophageal fistula (TOF) are part of a spectrum of conditions where there is defective embryological development of the trachea and oesophagus. The incidence is approximately 1 in 3,500 live births and many cases are diagnosed antenatally.

Can you describe the anatomical abnormality in more detail?

There are several patterns of abnormal anatomy (Figure 2.9.6).

It is worth being able to sketch these as it makes remembering and explaining the differences easier.

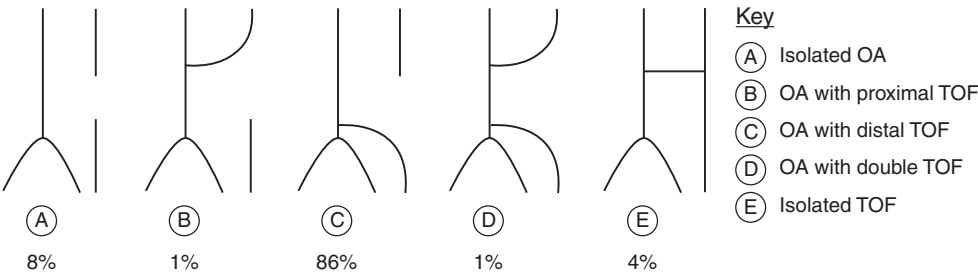


Figure 2.9.6 Types of trachea-oesophageal fistula.

The most common type involves a blind ending upper oesophagus with a fistula between the distal portion of the oesophagus and the trachea. This occurs in over 80% of cases.

What would you look for in your preoperative assessment of this infant?

It is important to look for other congenital abnormalities because 50% of infants with a TOF will have associated problems; these may be multisystem-associated conditions such as VACTERL. It is particularly important to look for evidence of congenital cardiac problems. The baby may have been born prematurely, raising the possibility of lung disease, retinal problems and impaired blood sugar regulation.

What is the VACTERL association?

If you are doing particularly well up to this point this may be asked.

This is an acronym used to describe a group of congenital problems which can occur together. An affected infant has vertebral, anorectal, cardiac, tracheo-oesophageal, renal and limb abnormalities.

How does tracheo-oesophageal fistula present?

Due to the defect between the oesophagus and trachea, the baby may have repeated episodes of coughing, choking and cyanosis secondary to soiling of the airway from secretions. This is exacerbated by attempts at feeding. It may also be detected by the presence of resistance to passage of an orogastric tube.

How should the infant be managed preoperatively?

The child cannot take oral fluids or nutrition so intravenous fluids should be administered taking care to avoid hypoglycaemia. A specialised suction tube called a Replogle tube can be used to prevent pooling of secretions in the upper part of the oesophagus. This works by letting air in to avoid high negative pressures sucking the oesophageal mucosa into the catheter.

What is the optimum timing of corrective surgery after birth?

In an otherwise uncomplicated case of TOF or OA, surgery should be carried out within 24 hours of delivery because accumulation of fluid in the upper oesophagus leads to a risk of aspiration.

How would you induce anaesthesia in this patient?

The tube in the upper pouch should be suctioned to reduce secretion load in the airway and oesophagus. Inhalational induction with sevoflurane is often the induction method carried out as mask ventilation increases the risk of gastric distention. Following an appropriate dose of a non-depolarising muscle relaxant the trachea is intubated with an appropriately sized endotracheal tube. Vigorous bag and mask ventilation is avoided because this can force air through the fistula from the trachea to the distal oesophagus and stomach. This can splint the diaphragm and impair ventilation. The infant is allowed to begin to breathe spontaneously on the tube at which point the tube can be removed to

permit examination of the airway with a rigid bronchoscope; inhalational anaesthesia can be maintained via the bronchoscope if required. The exact anatomy of the TOF can be established. The endotracheal tube can then be replaced and positioned so that the TOF is occluded. In this way the escape of gas from the trachea to the distal oesophagus and stomach during positive pressure ventilation is minimised.

What are the intraoperative concerns?

Specific patient factors include vigilance about other coexisting conditions, most importantly cardiac conditions. Intraoperative concerns for this case relate to positioning, monitoring, temperature regulation and maintenance of reliable intravenous access. The infant is usually positioned right lateral and strict attention must be paid to pressure areas. Invasive monitoring with arterial and central venous lines may be appropriate. Active warming methods must be used.

How will you manage the child postoperatively?

Most infants remain ventilated postoperatively and are cared for on the neonatal or paediatric intensive care unit. Appropriate sedation and analgesia should be ensured.

What can you tell me about congenital diaphragmatic hernia?

Congenital diaphragmatic hernia occurs in approximately 1 in every 3,000–4,000 live births. The diaphragmatic defect is most commonly left-sided and associated with a variable degree of lung hypoplasia; 40% of patients have significant associated congenital abnormalities, of which cardiac anomalies are most common. Experimental data suggests that abnormal lung development begins at an early stage of embryonic development, before the diaphragm begins to form. Subsequent diaphragmatic development is disturbed leading to the observed anatomical defect. Herniation of abdominal contents through the defect in the later stages of fetal development exerts a pressure effect, further impairing lung development. The affected lung has poorly developed airways, reduced numbers of type II pneumocytes and abnormal, highly reactive pulmonary vasculature. All affected infants have some degree of pulmonary hypertension.

Is surgical correction of the defect an emergency procedure?

No, the condition of the child should be optimised prior to considering a surgical procedure. Surgery does not improve gas exchange because of the associated pulmonary hypertension and lung hypoplasia secondary to the underdeveloped lung.

How should the patient be optimised prior to theatre?

Delivery should take place as close to term as possible to maximise lung development. Bag and mask ventilation should be minimised to prevent distension of the intrathoracic bowel. Intubation and ventilation with close attention to avoiding barotrauma should follow. The bowel lying within the chest cavity can be decompressed with an orogastric tube. The child should be transferred to a paediatric critical care area for invasive monitoring and further stabilisation. Appropriate investigations include arterial blood gases, chest X-ray and echocardiogram.

The principles of ventilating the infant are to achieve adequate gas exchange while preventing iatrogenic injury to the lung. Inspiratory pressures are limited and permissive hypercapnia is used. Inhaled nitric oxide may be trialled in patients with significant pulmonary hypertension although the evidence of an improved outcome in this condition is so far lacking.

Surgery is generally delayed for 24–48 hours to allow a fall in pulmonary vascular resistance.

What is exomphalos?

Exomphalos is a congenital defect of the abdominal wall. There is herniation of abdominal contents with a covering sac through a midline defect. It is caused by failure of the gut to migrate into the abdominal cavity during fetal development.

How does gastroschisis differ?

Gastroschisis is also an anterior abdominal wall defect allowing herniation of intra-abdominal contents. It does not occur in the midline and there is no covering sac over the exposed bowel.

Exomphalos is commonly associated with other congenital problems, particularly cardiac defects while gastroschisis is usually an isolated problem.

What are the problems associated with the two conditions?

There can be significant loss of heat and moisture by evaporation from the exposed abdominal contents. The bowel can be damaged by the drying effect and is a potential route for the entry of infection.

How is it managed?

The exposed bowel should be initially covered with a non-porous material such as cling film. Strict attention should be given to estimating and replacing fluid losses and to maintenance of normothermia. Bowel decompression is required prior to closure.

Primary surgical closure is attempted once the infant has been adequately resuscitated, emergency repair is only indicated if injury to the bowel is evident. Staged closure may be necessary if the abdominal contents cannot be reduced without compromising respiratory function; in this situation the exteriorised contents can be placed in a gradual decompression sling.

Further Reading

- Al Rawi O, Booker P. Oesophageal atresia and tracheo-oesophageal fistula *British Journal of Anaesthesia Continuing Education in Anaesthesia, Critical Care and Pain*. 2007; 7 (1) 15–19.
- Broemling N, Campbell F. Anaesthetic management of congenital tracheo-oesophageal fistula. *Paediatric Anaesthesia*. 2011; (21)1092–1099.
- King H, Booker P. Congenital diaphragmatic hernia in the neonate. *British Journal of Anaesthesia Continuing Education in Anaesthesia, Critical Care and Pain*. 2005; 5 (5) p171–174.
- Poddar R, Hartley L. Exomphalos and gastroschisis. *British Journal of Anaesthesia Continuing Education in Anaesthesia, Critical Care and Pain*. 2009; 9(2) 48–51.

2.9.7 Child Protection and Non-accidental Injury – Nicholas Marsden

This topic is one that is likely to be unfamiliar to most anaesthetists in training with few people encountering real life examples that they can use to remember the key aspects of the topic. It is an emotive topic, and may be difficult for some to talk about. It is most likely to be encountered as a written question, but may appear in the SOE. Having a structure to your answer will help to cover the key points that demonstrate an understanding of the issues. Remember that the safety of the child is the most important issue, greater than any other duty.

An 18-month-old child presents on the trauma list for the intramedullary nailing of a femoral shaft fracture. On review the child seems withdrawn but the parents suggest that she is a shy child who does not like strangers. The parents report that the child has had symptoms of a lower respiratory tract infection for two weeks and on examination there is a hand shaped bruise to the child's back.

What is child protection?

Children form a particularly vulnerable patient group, due to their dependence on others to provide their basic daily needs. Some patients are entirely dependent on others, most frequently their parents, to care for them and make decisions for them, whereas other paediatric patients may have more autonomy owing to their age, development or maturity, yet they still possess vulnerability to exploitation or maltreatment. Child protection aims to *identify* those patients that may be at risk of exploitation or maltreatment, *protect* those children that are being maltreated and *prevent* further maltreatment from occurring.

How can you classify forms of maltreatment?

Maltreatment of children can occur in different ways, on their own or in combination.

- Maltreatment, which can be further subclassified into:
 - Physical
 - Psychological
 - Sexual
 - Emotional
- Neglect
 - The failure to provide a child with their basic emotional and physical needs
- Exploitation
 - The use of a child to the advantage of another person
- Violence
 - May be orchestrated by an individual, groups or even the state

What are the risk factors for maltreatment?

The United Nations Convention of the Rights of the Child (UNCRC) recognises the distinct vulnerability of children owing to their distinct physical and cognitive

characteristics. These risk factors may be individual or environmental, which in turn may be dynamic as the child develops e.g., physical maturity or fixed e.g., ethnicity.

Individual factors include:

- Disability
 - Children with disabilities encompass a broad group, with a variety of capabilities and needs. Disability may be caused by physical or intellectual impairments and may form part of a chronic condition. They are particularly vulnerable to neglect.
- Mental health difficulties
 - The incidence of mental health difficulties in children is increasing and those from lower socioeconomic standing are 2–3 times more likely to develop problems and much less likely to receive help.
- Socioeconomic status
 - Poverty, poor housing and a poor neighbourhood are factors that increase a child's vulnerability. It should be noted, however, that higher socioeconomic standing does not mean that maltreatment does not feature in these communities and if there is clinical suspicion it should not be ignored.
- Out-of-home care
 - Outcomes for children that have been in out-of-home care are lower across education, health and employment.

Environmental factors include:

- Parental educational level
 - This can have a direct effect on a child's attainment, with children coming from a higher socioeconomic group being more likely to have higher level of education qualification, independent of the child's ability or skill.
- Parental health and health behaviours
 - Poor parental health may be transmitted to children, particularly through poor health behaviours, including diet, physical activity level, alcohol consumption, drug taking and smoking.
- Parental partner violence
 - Households suffering from parental partner violence are likely to contain a child under 5 years old, and co-occurrence with violence or maltreatment towards the child is encountered.
- Schooling/education
 - Early childhood care and education can have a protective effect on children, yet children from lower socioeconomic groups are half as likely to access services as those from higher socioeconomic groups.
- Neighbourhood
 - The neighbourhood that a child grows up in may reduce or increase the risk of maltreatment occurring. Neighbourhoods may provide a supportive network that improves the outcomes of children or concentrate negative factors such as poor housing, poverty and crime.

When might an anaesthetist encounter child protection issues?

All doctors are required to complete safeguarding training up to level 2 as we form part of a multidisciplinary team that looks after children. If there are concerns they should always be documented and raised with the local safeguarding team. Child protection issues may be encountered at any point along the clinical course. We may encounter injuries as part of the multidisciplinary team receiving critically ill patients in the emergency department, during a preoperative or pain clinic or routine perioperative care. Regardless of the situation, the basic principles of management remain the same.

NICE has provided guidance for when to 'consider' and 'suspect' maltreatment: consider when maltreatment may form part of the differential diagnosis; suspect is a more serious level of concern that maltreatment has taken place but does not constitute proof.

What clinical presentations may suggest child maltreatment?

Anaesthetists may encounter children in many different clinical scenarios, and should be vigilant to the possibility of maltreatment as it represents an opportunity to intervene. Always consider maltreatment when the explanation for the presentation appears inadequate or a medical explanation cannot be found. When engaging in communication with a child, the behaviour and emotional state should be considered. Children that appear fearful, withdrawn, aggressive or indiscriminately affectionate, which is inconsistent with their developmental stage or cannot be explained by medical causes may be suggestive of maltreatment.

Clinical scenarios that should raise suspicion include:

- Repeated apparent life-threatening events especially if they are witnessed by only one parent or carer.
- Life-threatening event with bleeding from the nose or mouth without medical explanation
- Repeated presentations of ingestion of inappropriate substances
- Deliberate administration of prescribed or non-prescribed drugs
- Unexpected blood levels of drugs not prescribed for the child
- Children presenting with hyponatraemia without medical explanation
- Non-fatal submersion injury where the explanation is unsuitable or suggests a lack of supervision
- Unusual patterns of presentation to medical services including frequent attendance
- Fabricated or induced illness e.g., the history, assessment and investigations do not fit with a recognised clinical picture.

Features of neglect include children with severe or persistent infestations e.g., head-lice, those whose clothing and footwear are inappropriate, malnutrition or if parents or carers do not administer prescribed medication or fail to engage with child health promotion programmes including immunisation.

What is meant by the term non-accidental injury (NAI)

Non-accidental injury is a term that is used to describe injuries that are unusual for the child's age or development or the mechanism of injury. When seeking an explanation

for an injury it is important to consider whether it is one or a combination of the following:

- Implausible
- Inadequate
- Inconsistent.

What physical features might suggest NAI?

NAI should be considered if the child presents with injuries that are unexpected for a child of that age or the explanation of cause does not fit with the injury. Examples include:

- Bruises
 - Consideration should be given to NAI if the bruising is in the shape of a hand, implement, teeth marks or grip
 - If the bruising cannot be explained by a medical condition and the explanation is implausible e.g., bruising in a child who is not independently mobile or bruising on the non-bony parts of the body and face including eyes, ears and buttocks.
- Bites
 - Both human and non-human may alert a clinician to maltreatment. If the appearance of the bite mark does not fit with being caused by a small child or if there is evidence of animal bites then physical abuse or neglect should be considered.
- Lacerations
 - Lacerations that appear to be circumferential, caused by ligatures, occur in a child who is not independently mobile or are in an area usually protected by clothing are all features of NAI.
- Thermal injuries
 - Both hot and cold thermal injuries may be the result of NAI and should be considered if the child is not independently mobile, is in the shape of an implement e.g., a cigarette, imply immersion e.g., glove and stocking distribution of a scald injury or the child presents with hypothermia with no suitable explanation.
- Fractures
 - In the absence of a medical condition predisposing to fragile bones, NAI should be considered if the explanation for the fracture is unsuitable or if there is evidence of fractures of different ages or X-ray evidence of fractures that were not clinically evident.
- Intracranial injuries
 - In the absence of accidental major trauma, intracranial injury where the child is under 3 years of age, or evidence of retinal haemorrhage, or with rib or long bone fractures NAI should be considered.
 - Multiple subdural haemorrhages with or without hypoxic brain injury is also suggestive of NAI.

- Eye/spinal/visceral/oral injuries
 - In the absence of accidental major trauma, injuries to eyes such as retinal haemorrhages, or injury to the vertebrae or spinal canal, or intrathoracic or intra-abdominal injury with an unsuitable explanation may suggest NAI.
- Ano-genital injury
 - Sexual abuse may be apparent through injuries to the genital, anal or perianal area, which may present as bruising, swelling, laceration or discharge. Foreign bodies in the vagina or anus may also indicate sexual abuse.
 - Any child under the age of 13 who presents with either a sexually transmitted infection or pregnancy should warrant further investigation into sexual abuse.
 - If a young woman aged 16 or 17 years presents with pregnancy consider sexual abuse if:
 - There is a clear difference in power or mental capacity between the mother and putative father, particularly if the relationship is incestuous, or with a person in a position of trust e.g., teacher.
 - Or there is concern that the young woman may be being exploited or that the sexual activity was not consensual.

What steps should you take if you suspect NAI?

When NAI is suspected or there are safeguarding concerns, discussion should take place with the safeguarding team or on-call paediatric consultant if out of hours. If the concerns are raised while the child is under general anaesthetic, the anaesthetic should not be prolonged while advice is sought, and it should be remembered that consent has not been sought for examination related to the concerns raised. All members of the theatre team should be able to raise concerns about child welfare, and the concerns should be investigated appropriately.

Once concerns are raised the safeguarding team or consultant paediatrician will involve the parents/carers and the child in question in a consultation where a history from the child and parents/carers is sought along with an examination, which may result in a reasonable explanation being put forward.

Consideration for a photographic record or forensic sampling, along with involvement of other agencies such as social services or the police may happen following the initial consultation. A decision will also be made as to whether the child will be allowed home at the normal time following their anaesthetic.

Concerns must be recorded contemporaneously in the clinical record, with an accurate record of what has been observed and heard from whom and when. A record of why this is of concern should be made. At this point maltreatment as a differential diagnosis is either considered, suspected or excluded.

Further Reading

When to suspect child maltreatment (CG89).
NICE, London 2017

The United Nations Convention on the Rights of the Child. UNICEF, London 1989

Perioperative Medicine

2.10.1 Cardiopulmonary Exercise Testing – Niladri Das

What is cardiopulmonary exercise testing?

Cardiopulmonary exercise testing (CPET) is a non-invasive, dynamic assessment of the cardiopulmonary system at rest and during exercise. It provides an objective assessment of an individual's functional capacity, that is the ability to respond to the increased metabolic demand associated with major surgery. This information can help to estimate the risk of perioperative morbidity and mortality as well as assist clinicians in the decision-making process between surgical and non-surgical management of the patient. The outcome of this preoperative investigation may also inform surgical consent, planning and optimisation of a patient in preparation for their operation.

How is CPET performed?

Cardiopulmonary exercise testing is normally conducted on a cycle ergometer with each test taking approximately 10 minutes in duration. Data is collected from a patient's expired gases using a rapid gas analyser and pressure differential pneumotachograph simultaneously with continuous 12-lead ECG, oxygen saturation and non-invasive blood pressure monitoring. Measurements are taken at rest, during unloaded cycling (pedalling without resistance), loaded cycling (pedalling against an increasing resistance at a predetermined ramp rate) and in the recovery phase after exercise. Key measurements include the following: work rate (in Watts), oxygen consumption (VO_2), carbon dioxide production (VCO_2), respiratory exchange ratio (RER), tidal volume (V_T), respiratory rate, ventilatory equivalents for oxygen ($\text{V}_\text{E}/\text{VO}_2$) and carbon dioxide ($\text{V}_\text{E}/\text{VCO}_2$). Demographic data to include the patients age, gender, height and weight can be inputted into the computer and predicted normal values can be calculated. The data points are represented graphically in a format called the nine-panel plot. It is important to calibrate the machine prior to each exercise test. During the evaluation period, the patient is able to communicate with the clinician through previously agreed signs while the severity of symptoms such as chest pain, difficulty breathing and leg pain can be assessed using pointing charts such as the Borg scale. Although a relatively safe and reliable investigation, the test can be terminated at any point in the event of clinically inappropriate symptoms or complications.

What are the contraindications to CPET?

Absolute contraindications include recent acute myocardial infarction, unstable angina, severe symptomatic aortic stenosis, poorly controlled heart failure, uncontrolled arrhythmias and active endocarditis. Relative contraindications include asymptomatic severe aortic stenosis, untreated severe hypertension (>200 mmHg systolic), hypertrophic cardiomyopathy, significant pulmonary hypertension, untreated lower extremity thrombosis or pulmonary embolus, abdominal aortic aneurysm, electrolyte abnormalities and advanced pregnancy. Patients with relative contraindications should be directly supervised by a doctor and the risks versus benefits should be discussed on an individual case basis. CPET is a dynamic assessment and the test can be terminated early if the risk-benefit relationship were to change during the evaluation.

Briefly describe how you would interpret the nine-panel plot.

The panels of a nine-panel plot are numbered 1–9 from top left to bottom right. The cardiovascular system is represented by panels 2, 3 and 5 whereas the ventilation is represented by panels 1, 4 and 7. Panels 6, 8 and 9 demonstrate ventilation-perfusion relationships. The Perioperative Exercise Testing and Training Society (POETTS) provide guidelines on a standardised approach to reporting CPET tests.

A series of nine questions should be asked by the investigator. Firstly, one must determine whether the test was maximal in terms of effort, that is, will the CPET derived values of anaerobic threshold and peak VO_2 be valid. Maximal effort is defined as either achieving more than 80% of the predicted work in panel 3, a heart rate >80% of the predicted maximum in panel 2 or an RER of greater than 1.15 as seen in panel 8. Subsequently identify the peak VO_2 from panel 3 and establish whether the VO_2 /work relationship was normal. Typically, as resistance increases on the cycle ergometer, so too does the individual's oxygen consumption. Next, determine the anaerobic threshold from panel 5 using the 'V slope method'. This is the point at which VCO_2 increases disproportionately when compared with VO_2 . Note the VO_2 at the point of intersection and calculate the anaerobic threshold based on the patient's weight. Referring back to panel 2, determine whether there was a normal heart rate response to increasing exercise intensity. Oxygen pulse is seen in panel 2. This is the VO_2 divided by heart rate and can be used as a surrogate for stroke volume. Thus, oxygen pulse should increase at the beginning of exercise reaching a plateau at its highest predicted value. The clinician should note any ECG changes as a result of exercise. Finally, one must assess whether there was any ventilatory limitation. The normal response to exercise follows a linear increase in ventilation until the anaerobic threshold is reached at which point there is a disproportionate increase in ventilation driven by CO_2 production. In a healthy subject, maximum ventilation does not exceed 80% of the maximum voluntary ventilation (MVV) which is a measure of the maximum volume of air that can be inhaled and exhaled within 1 minute.

Explain the terms anaerobic threshold and peak VO_2 in addition to relevance in clinical practice?

The anaerobic threshold is a metabolic rate defined as the VO_2 above which arterial lactate first begins to increase during incremental exercise. It is a marker of exercise

capacity, expressed in ml/kg/ min, and cannot be influenced by patient volition. As an individual continues to exercise, the oxygen demand will begin to exceed supply. Muscle cells will generate ATP anaerobically and produce lactic acid. This is buffered by circulating bicarbonate and gives rise to an increase in CO₂ production. The VO₂ at the point at which this occurs is called the anaerobic threshold and can be derived from plots 3–6, 8 and 9 of the nine-panel plot. At the anaerobic threshold, the slope of increasing VCO₂ curve exceeds that of the VO₂ curve and the RER will increase above 1. As compared with other CPET variables, the anaerobic threshold can predict postoperative morbidity and mortality in a wide range of surgical populations.

Peak VO₂ is a metabolic rate defined as the highest VO₂ achieved on a rapid incremental test at the end of exercise. It is calculated as an average value over 20 seconds and reflects the patient's best effort. The reported value, in ml kg⁻¹ min⁻¹, correlates well with postoperative morbidity and mortality in the surgical population. The maximum VO₂ that can physiologically be attained by a patient is defined as the oxygen uptake during an exercise intensity at which actual oxygen uptake reaches a maximum beyond which no increase in effort can increase it. This can be demonstrated with a plateau in VO₂ with increasing work load.

CPET, in addition to providing a detailed assessment of a patient's functional status, is a strong diagnostic tool for numerous medical conditions to include ischaemic heart disease, heart failure, obstructive and restrictive lung disease. Major cavity surgery is associated with a significant increase in oxygen consumption and is sustained for several days postoperatively. Hence, patients require a substantial cardiac and respiratory physiological reserve. CPET represents a non-invasive simulation of the requirements for major surgery and may facilitate decision-making on postoperative destination. An anaerobic threshold of 11 ml/kg/min and peak VO₂ of 20 ml/kg/min is typically required for major surgery while those patients unable to reach this figure will likely benefit from a critical care bed postoperatively. Advances in prehabilitation and exercise training can improve an individual's cardiopulmonary fitness and possibly reduce postoperative mortality.

What do you understand by the term metabolic equivalent of tasks?

One metabolic equivalent of tasks (MET) represents the oxygen consumption of an adult at rest (~3.5 ml kg/kg/min). The number of METs are assigned according to the degree of exercise undertaken.

What is the Duke Activity Status Index?

The Duke Activity Status Index (DASI) is a patient questionnaire comprising of 12 questions used to assess an individual's ability to perform activities of daily living. The total score can be used as a surrogate for peak oxygen consumption. The subjective nature of this assessment tool is a limitation to its use.

What is a functional walk test?

Functional walk tests are a relatively inexpensive alternative to CPET when assessing functional capacity in an individual. The 6-minute walk test (6MWT) and incremental shuttle walk test (ISWT) are most commonly used. The former measures how far an individual can walk along a flat corridor, turning around cones at each end, at a normal

pace in 6 minutes. Typical distances achieved by healthy subjects are in the region of 500 metres. Additional measurements include oxygen saturations, heart rate and the Borg scale assessment for dyspnoea and leg fatigue. In contrast, the ISWT involves patients walking at speeds that increase every minute by 0.17 m/s in time to audio signals. Failure to reach the cone prior to the next audio signal or exhaustion terminates the test. Distances walked in both assessments correlate well with peak oxygen consumption and maximum work capacity.

Further Reading

Agnew N. Preoperative cardiopulmonary exercise testing. *BJA Education*. 2010; 10: 33–37.

Chambers DJ, Wisely NA. Cardiopulmonary exercise testing: A beginner's guide to the 9-panel plot. *BJA Education*. 2019; 19: 158–164.

Levett DJH, Jack S, Swart M, et al. Perioperative cardiopulmonary exercise testing (CPET): Consensus clinical guidelines on indications, organisation, conduct, and physiological interpretation. *British Journal of Anaesthesia*. 2018; 120: 484–500.

2.10.2 Preoperative Assessment and Prehabilitation – Joseph Swani

What are the roles of a preoperative anaesthetic assessment service?

- Ensure patients are fully informed about the procedure and interventions to be taken.
- Estimate the level of risk for the patient and ensure that the patient understands their individualised risk so that they can make informed decisions about their care.
- Provide patients with an opportunity to discuss choices of anaesthetic technique, pain relief options and risks.
- Identify comorbidities and consider what further investigations and assessments may be needed, whether optimisation of comorbidities may be possible and balance this against the urgency of the operation.
- Assess suitability for day surgery.
- Provide specific information on which drugs to continue or discontinue perioperatively and provide fasting guidance.
- Identify patients who are high risk and advise on appropriate postoperative levels of care.
- Planning discharge after surgery.

What are the potential benefits of a preoperative anaesthetic assessment service?

There may be reduced complication rates and mortality, improved patient satisfaction and a decrease in cancellations on the day of surgery.

What team members are involved in preoperative assessment?

These include anaesthetists, surgeons, physicians and general practitioners. In addition, specialist anaesthetic preoperative assessment nurses are an integral part of the

preoperative assessment team. Physician Associates (Anaesthetics) may also have a role in some trusts.

Why are patients risk assessed prior to surgery?

Estimating risk from a surgical procedure allows better shared decision-making with the patient and informed consent. In addition, it provides a measure which can help aid decisions on perioperative optimisation and the planning of intraoperative and postoperative care.

What risk-assessment tools are commonly used in anaesthesia?

These can be divided into risk scores (Table 2.10.2.1) and risk prediction models (Table 2.10.2.2). Risk scores allocate a weighting to independent outcome predictors, then often sum these weightings to give a risk score which is used to place patients on a scale and compare risk to others. Risk prediction models aim to provide a more individualised risk prediction by entering patient data into a multivariable risk prediction model. They are more complex to use but more accurate for an individualised risk prediction.

Table 2.10.2.1 Common risk scores

ASA physical status score	<ul style="list-style-type: none"> • Categorises patients into six subgroups (normal healthy patient, mild systemic disease, severe systemic disease, severe systemic disease that is a constant threat to life, moribund patient who is not expected to survive without the operation, brain-dead patient for organ donation). • High ASA score is predictive of increased postoperative complication and mortality.
Lee's Revised Cardiac Risk Index (RCRI)	<ul style="list-style-type: none"> • Most commonly used risk score for estimating risk of cardiac complications from non-cardiac surgery. • Six independent predictors (high-risk surgery, ischaemic heart disease, congestive cardiac failure, cerebrovascular disease, insulin therapy for diabetes and preoperative creatinine >176). • One point for each predictor, sum of score is the Lee class and provides a predictor of cardiac complications.
Assess respiratory risk in surgical patients in Catalonia (ARISCAT)	<ul style="list-style-type: none"> • Used for predicting postoperative pulmonary complications. • Seven variables (age, preoperative SpO₂, respiratory infection in the last month, preoperative anaemia, surgical incision site, duration of surgery, emergency procedure). • Categorises patients into low, intermediate and high-risk groups.

Table 2.10.2.2 Common risk prediction models

Portsmouth Physiological and Operative Severity Score for the enUmeration of Mortality and Morbidity (P-POSSUM)	<ul style="list-style-type: none"> 12 physiological variables and 6 surgical variables used to predict 30-day postoperative mortality.
National Emergency Laparotomy Audit (NELA) calculator	<ul style="list-style-type: none"> Much of the same data as P-POSSUM. Used to predict 30-day postoperative mortality. Differences to P-POSSUM are that NELA includes ASA, gender and creatinine whereas P-POSSUM includes haemoglobin. NELA uses more continuous variables and there is less weight given to operative severity. NELA is more accurate and specific for emergency laparotomy.
Surgical Outcome Risk Tool (SORT)	<ul style="list-style-type: none"> Estimates 30-day mortality. The updated second version includes a question on the clinician's estimate of risk and unlike the original SORT model, can be used for a wider variety of surgery including neurological and cardiothoracic surgery.
American College of Surgeons national surgical quality improvement project (ACS NSQIP) universal surgical risk calculator	<ul style="list-style-type: none"> Uses 21 preoperative factors to predict a multitude of outcomes within 30 days of surgery. Not only provides risk prediction for mortality but also for a number of complications including pneumonia, cardiac complication, surgical site infection, urinary tract infection venous thromboembolism, renal failure, readmission, return to theatre and discharge to a nursing or rehab facility.

What is prehabilitation?

Prehabilitation is a multicomponent process which aims to enhance a patient's functional capacity prior to surgery in order to help them withstand the impact of the upcoming surgical event. Prehabilitation focusses on the preoperative phase; this is in contrast to 'rehabilitation' which is focussed on postoperative care.

What are the benefits of prehabilitation?

Impaired preoperative functional capacity is strongly associated with poor surgical outcome. Improving functional capacity through a prehabilitation programme may have benefits which include reduced length of stay, postoperative pain and postoperative complications.

What are the components of a prehabilitation programme?

A prehabilitation programme runs in the time period between a decision to operate and the surgery itself. The interventions are best provided with a multidisciplinary, multi-modal approach. The team can include anaesthetists, surgeons, medical physicians,

physiotherapists, nutritionists and psychologists. Firstly, basic measurements such as weight, height and percentage fat are recorded. Functional capacity, nutritional status and mood are assessed and these are monitored throughout. Component areas of focus during a prehabilitation programme are medical optimisation, physical exercise, nutritional support and psychological support.

Medical optimisation:

- Lifestyle modification through smoking cessation, reducing alcohol intake and weight management.
- Optimisation of medical conditions including anaemia, hypertension, diabetes, respiratory disease and heart disease. This involves a review of pharmacological therapy.

Physical exercise

- Exercise capacity can be assessed for a baseline and can also be used to provide objective assessment of an improvement in fitness. The most reliable form of testing is cardiopulmonary exercise testing (CPET); however the 6 min walk test is simpler to perform.
- Exercise programmes vary in their duration and frequency. Strength and aerobic exercises in combination can reduce the level of decline in muscle strength and cardiorespiratory fitness postoperatively. Programmes benefit from being clear and structured, facilitating an environment which encourages engagement in the process.

Nutritional support

- Good preoperative nutritional status improves postoperative outcomes.
- Poor nutritional status is associated with impaired immunity and wound healing and malnutrition in patients attending for surgery is often due to the disease process itself.
- Patients should be formally assessed for nutritional risk prior to major surgery.
- Optimisation of nutrition should start as soon as possible preoperatively.
- Clear fluid carbohydrate drinks in the immediate preoperative period can reduce insulin resistance, improve patient comfort (reduced thirst, hunger, dehydration, headache and nausea), minimise protein losses and improve postoperative muscle function.

Psychological support

- Psychosocial stress impacts on physiology and can lead to immunological dysfunction and worsened functional recovery.
- Psychological support aims to reduce the psychological distress associated with a diagnosis and surgery, as well as enhance motivation and compliance with other parts of the prehabilitation programme such as exercise and nutritional programmes.

What are the potential physiological benefits of a prehabilitation exercise programme?

Cardiorespiratory fitness has an impact on morbidity and mortality after surgery. Those with poor cardiorespiratory reserve are more likely to have complications following

surgery. Surgical stress results in neuroendocrine, metabolic and immunological changes which increase oxygen consumption, metabolic rate and protein catabolism. Following surgery, patients are likely to be less active physically which can result in a fall in functional capacity. This includes general deconditioning with muscle atrophy and loss of muscle strength as well as cardiac deconditioning with a reduction in VO_2max , stroke volume and cardiac output. The aim of increasing functional capacity before an operation is to prepare the body for the metabolic impact of surgery and the recovery period. Exercise training leads to an increase in cardiac output, arteriovenous oxygen difference and VO_2max . Skeletal muscles increase mitochondrial content and overall capacity for oxygen uptake. Functional reserve is increased, enhancing the body's ability to withstand the stress of surgery and the impact of the recovery period.

Further Reading

Association of Anaesthetists of Great Britain and Ireland. Pre-operative Assessment and Patient Preparation The Role of The Anaesthetist. *AAGBI Safety Guideline* 2010. <https://anaesthetists.org/Home/Resources-publications/Guidelines/Pre-operative-assessment-and-patient-preparation-the-role-of-the-anaesthetist-2/>.

Banugo P, Amoako D. Prehabilitation. *British Journal of Anaesthesia*. 2017; 17: 401–405.

Fawcett W, Ljungqvist O. Starvation, carbohydrate loading, and outcome after major surgery. *British Journal of Anaesthesia*. 2017; 17: 312–316.

Schonborn JL, Anderson H. Perioperative medicine: A changing model of care. *British Journal of Anaesthesia*. 2019; 19: 27–33.

Stones J, Yates D. Clinical risk assessment tools in anaesthesia. *British Journal of Anaesthesia*. 2019; 19: 47–53.

Wong DJN, Harris S, Sahni A, et al. Developing and validating subjective and objective risk-assessment measures for predicting mortality after major surgery: An international prospective cohort study. *PLoS Med*. 2020; 17: e1003253.

Yentis S, Hirsch N, Ip J. 2019. *Anaesthesia, Intensive Care and Perioperative Medicine A-Z*. 6th ed. Elsevier.

2.10.3 Anaesthetic Considerations for Cancer Surgery – Matthew Townsend

As an anaesthetist you will encounter patients at all stages of their cancer journey. You will be expected to demonstrate familiarity with assessment and management of these patients, as well as some knowledge of how perioperative care may affect longer term outcomes.

You are in the anaesthetic pre-assessment clinic and see a 65-year-old man with stage 3 rectal adenocarcinoma listed for an abdominoperineal resection.

What is a cancer staging system?

It is used to describe the extent and spread of cancer. A commonly used system is the TNM staging system. ‘T’ refers to the primary tumour, ‘N’ for involvement of nearby lymph nodes and ‘M’ for distant metastases. Numbers are assigned after each letter to describe increasing size, extent or spread. Another system uses stages 0–4. Stage 0 refers to cancer in situ and stage 4 is metastatic disease. Stage 3, as in this case, refers to a tumour that has grown locally invasive and spread to lymph nodes, but has not metastasised more distantly.

What risk factors do you know for developing bowel cancer?

Increasing age, male sex, smoking, alcohol, obesity, family history and a diet high in red and processed meats are all risk factors for developing bowel cancer. There is a higher incidence in people with inflammatory bowel diseases such as Crohn's and ulcerative colitis. There are also some rare genetic predispositions for colorectal cancers such as familial adenomatous polyposis (FAP) and hereditary non-polyposis colorectal cancer (HNPCC).

The patient tells you that he was diagnosed following a screening test.

What do you know about bowel cancer screening?

Currently, bowel cancer screening is offered to men and women between the ages of 60 and 74. They are offered screening every two years and are sent a home testing kit called the faecal immunochemical test which detects small amounts of blood in the stool. Positive tests are then invited to undergo colonoscopy. This programme is gradually being expanded to include all people aged 50 to 74.

Do you know of any other screening programmes in the UK?

Cervical screening for human papillomavirus is offered to all women between the ages of 25 and 64. Breast screening with mammography is offered to women between the ages of 50 and 71. There are also non-cancer screening programmes such as those for abdominal aortic aneurysms.

Going back to this case, can you describe your preoperative assessment of this patient?

This question should be answered using a structured approach to history, examination and investigations. Specific points for this case will include eliciting any systemic effects of malignancy and anti-cancer treatments. Remember the effects of paraneoplastic syndromes associated with some tumour types e.g. Cushing's syndrome and SIADH (small cell lung cancer) and carcinoid syndrome (neuroendocrine tumours).

I would start by taking a full history from the patient including current medical conditions and any previous anaesthetic and surgical history. I would want to focus particularly on any symptoms related to his tumour or treatments he has received. He may be suffering the effects of anaemia, malnutrition, weight loss or dehydration. I would take a detailed drug history and ask specifically about neoadjuvant treatment and if he has had any side effects. Chemotherapy commonly causes nausea, vomiting and dehydration but can also cause toxicity to some organs. I would ask about symptoms such as shortness of breath, chest pain, palpitations and fever which would make me suspicious for toxicity. I would also ask about any other anti-cancer treatments he may have received such as radiotherapy or immunotherapy.

This man is planned to undergo a major intra-abdominal procedure and I would also ensure I took a full cardiorespiratory and functional history to assess his suitability.

My general examination may reveal pallor, cachexia or muscle wasting. I would like to know an accurate weight to ensure I am making safe and appropriate drug dosing. Further examination would be guided by any symptoms in the history suggestive of a

particular organ toxicity. I would like to perform a respiratory exam which may reveal wheeze or creptitations secondary to pulmonary damage, and a cardiovascular exam looking for signs of failure. If he has described any symptoms of peripheral neuropathy, I would like to perform and document a careful neurological examination, especially if he is to undergo any regional anaesthesia. If present, I would also want to investigate for potential central or autonomic involvement.

Investigations I would like for this patient include a full blood count, looking specifically for anaemia or myelosuppression secondary to chemotherapy. Urea and electrolytes might show evidence of kidney injury or electrolyte imbalance. Liver function tests and an ECG should also be ordered as these may reveal asymptomatic organ toxicity. Further investigations such as an echocardiogram, chest X-ray or pulmonary function tests may be indicated if specific toxicity is suspected.

You mentioned chemotherapy. Can you give any examples of drugs or side effects?

The examiner will not expect you to know specific regimes, but they will expect you to have some idea of types of drugs and their common side effects. An example or two of each will suffice.

Chemotherapy drugs work by targeting and destroying rapidly dividing cancer cells. They also damage non-cancerous cells, and this is what leads to unwanted organ damage.

Examples of chemotherapy agents include

- Alkylating agents e.g. cyclophosphamide, cisplatin
- Anti-metabolites e.g. methotrexate, 5-fluorouracil
- Anthracyclines e.g. doxorubicin, idarubicin
- Cytotoxic antibiotics e.g. bleomycin, mitomycin
- Vinca alkaloids e.g. vincristine.

Side effects common to almost all chemotherapeutic agents include gastrointestinal upset, myelosuppression and immunosuppression.

Serious side effects can be classified by the organ commonly affected. Pulmonary toxicity causing pneumonitis and fibrosis are seen with methotrexate and bleomycin. Patients may present with cough, dyspnoea or pleuritic chest pain. Cardiac toxicity caused by drugs such as doxorubicin can manifest as arrhythmia, myocarditis, pericarditis, cardiomyopathy or congestive cardiac failure. Renal toxicity is caused by many chemotherapy agents as they are often renally excreted. Cisplatin and cyclophosphamide are known to cause acute kidney injury and may only be picked up preoperatively by a decline in renal function. Hepatotoxicity is also a feature of many drugs, usually presenting as an asymptomatic transaminitis. Methotrexate is known to cause fatty liver, fibrosis and even cirrhosis. Neurotoxicity is notoriously associated with vincristine administration, and can present with neuropathy, autonomic dysfunction and seizures.

Do you know of any specific concerns with bleomycin?

Bleomycin is a glycopeptide antibiotic used in the treatment of lymphomas and germ cell tumours. It can cause sub-acute pulmonary damage, which can then be exacerbated by administering high concentration oxygen. Even short periods of hyperoxia, delivered

years after initial treatment, have been implicated in causing rapidly progressive pulmonary fibrosis. The British Thoracic Society recommend that unless the patient is hypoxic, oxygen administration should be avoided in anybody who has received bleomycin therapy. In emergency situations, oxygen should be used cautiously to target a saturation range 88–92%.

What would your anaesthetic plan be for this patient?

Many colorectal procedures are amenable to laparoscopic approaches and the consequences of pneumoperitoneum need to be considered in addition to usual airway, respiratory, cardiovascular and analgesic plans. An abdominoperineal resection will usually require the patient to be turned into a prone position during the case.

I would intubate this patient as this is long duration intra-abdominal surgery and there is a significant increased risk of reflux from pneumoperitoneum, steep Trendelenburg and prone positions. Raised intra-abdominal pressures will also affect ventilation with basal atelectasis, reduced FRC and V/Q matching, as well as increased airway pressures. The cardiovascular effects of laparoscopy include a reduction in preload and an increase in afterload. This can lead to a reduction in cardiac output at higher inflation pressures and can precipitate ventricular failure in susceptible patients. These effects are of particular importance if there are any preoperative concerns about cardiac toxicity caused by neoadjuvant treatment.

I would insert two large bore IV canulae in case of major blood loss and an arterial line to allow beat-to-beat monitoring and blood gas sampling. Venous access is often difficult after chemotherapy, and central venous cannulation is commonplace. These patients are prone to large fluid shifts as well as the haemodynamic consequence of pneumoperitoneum and steep head-down positioning. I would like to use a cardiac output monitor if available. My aim is to use goal-directed fluid therapy and aim for perioperative euvolaemia. They may be suitable to follow an enhanced recovery programme which minimises the effects of prolonged fasting and bowel preparation.

I would use a multimodal, opioid-sparing analgesic technique which includes regional anaesthesia. Spinal or epidural analgesia is likely to be beneficial, as are fascial plane blocks such as transversus abdominis plane (TAP) and rectus sheath blocks if there is a contraindication to neuraxial technique (e.g. thrombocytopenia).

I will pay particular attention to thermoregulation, positioning and pressure areas, especially when in steep Trendelenburg and prone positions. Myelosuppression can result in increased susceptibility to infection, and I would ensure I used strict aseptic technique in addition to appropriate antibiotic prophylaxis.

Why are cancer patients at an increased risk of perioperative venous thromboembolism?

These patients share common perioperative risk factors for thrombus formation including dehydration, immobility and initiation of the inflammatory stress response. In addition to this, cancer causes an upregulation in pro-coagulants such as tissue factor, and a reduction in levels of circulating anticoagulants such as antithrombin and protein C. Cellular damage caused by chemotherapy, immunotherapy and radiotherapy all produce pro-inflammatory responses, further increasing the risk of thrombosis.

What is tumour lysis syndrome?

This is a metabolic syndrome caused by the rapid death of cancer cells. Intracellular metabolites are released into the circulation causing dangerously high levels of phosphate, uric acid and potassium and hypocalcaemia. It is usually seen in patients with blood cancers following the start of chemotherapy. If untreated it can lead to acidosis, renal failure, cardiac arrhythmia, seizure and death.

Do you know any risk factors for tumour lysis syndrome?

There is a greater risk with increasing volume and speed of cell death. Risk factors include

- High tumour burden
- High grade tumours
- Increasing age
- Pre-existing renal impairment
- Treatment with highly active agents

Do you know of any potential links between anaesthetic technique and tumour recurrence?

This is an ever topical (and controversial) area of research and you might be asked to discuss some of the recent evidence. It would be worth keeping abreast of any recent publications in this area in the lead up to the exam.

Maintenance anaesthesia:

Volatile anaesthetics are potentially pro-carcinogenic. They are thought to cause an increase in a number of mediators which promote malignant cell growth, while also reducing levels of natural killer cells responsible for cell death. The overall result is an environment that supports tumour growth and spread. In contrast, propofol may in fact do the opposite – reducing tumour growth factors and preserving immune responses that lead to cancer cell apoptosis. While there are lab and retrospective studies which support this theory, there is not yet enough good quality clinical evidence to support TIVA over volatiles for cancer surgery.

Regional anaesthesia:

Surgical stress causes systemic immunosuppression, and this in turn allows tumour growth. Regional techniques, in addition to their analgesic benefit, are thought to attenuate the stress response and could therefore reduce tumour recurrence. While there have been several retrospective studies to support this, the largest randomised control trial conducted to date (Sessler et al. Lancet, 2019) failed to show any difference in rates of breast cancer recurrence between regional anaesthesia (propofol and paravertebral block) vs general anaesthesia (sevoflurane and opioid).

Opioids:

Blunting the surgical stress response with any form of analgesia is likely to be beneficial. However, high systemic doses of opioids have themselves been implicated in tumour recurrence. Mu opioid receptors are expressed on some cancer cells, and activation of

these can cause angiogenesis, invasion and migration. While evidence on cancer survival remains mixed, it would be pragmatic to use multimodal and opioid-sparing techniques when anaesthetising these patients.

Further Reading

British Thoracic Society Guideline for oxygen use in adults in healthcare and emergency settings. *Thorax*. 2017; 72:i1–i90.

Evans MT, Wigmore T, Kelliher LJ. The impact of anaesthetic technique upon outcome in oncological surgery. *British Journal of Anaesthesia Education*. 2019; 19 (1): 14–20.

Jones GL, Will A, Jackson GH, Webb NJ, Rule S. Guidelines for the management of tumour lysis syndrome in adults and children with haematological malignancies

on behalf of the British Committee for Standards in Haematology. *British Journal of Haematology*. 2015; 169(5): 661–671.

National Institute for Health and Clinical Excellence. Colorectal Cancer. 2020 www.nice.org.uk/guidance/ng151/chapter/Recommendations#management-of-local-disease

Sessler D, Pei L, Huang Y, Fleischmann E, Marhofer P, Kurz A, et al. Recurrence of breast cancer after regional or general anaesthesia: A randomised controlled trial. *The Lancet*. 2019; 394(10211): 1807–1815.

2.10.4 Anaesthesia for the Elderly – Joseph Swani

What are the anatomical and physiological changes in the older patient that are relevant to anaesthesia?

Cardiovascular:

- Significant cardiovascular disease may be present including ischaemic heart disease and valvular heart disease.
- With age there is an increase in fat infiltration and fibrosis of the cardiac conducting pathways, with a higher prevalence of cardiac conduction defects and arrhythmias including atrial fibrillation.
- Maximal cardiac output decreases and there is a reduced ability for the cardiovascular system to respond to stress. Reduced autonomic system function impairs the cardiovascular response to hypotension. Myocardial catecholamine receptors are downregulated, which reduces the effect of catecholamines and sympathomimetic agents. There is reduced ventricular contractility and stroke volume. Patients are less able to increase cardiac output in response to falls in systemic vascular resistance and fluid loss. Diminished baroreceptor response and reduced responsiveness to angiotensin II further impairs the ability to respond to hypovolaemia. Anaesthetic agents (IV and inhalational) are more likely to cause hypotension.
- Lower cardiac output results in a longer arm-to-brain circulation time for intravenous drugs, this means that the onset of anaesthesia following intravenous induction is delayed in comparison to younger patients.
- Arterial vessels become less compliant which increases systemic vascular resistance, widens pulse pressure and causes systolic hypertension. This may cause left ventricular strain and hypertrophy. Vasoconstrictors are less effective.
- There is increased capillary permeability, which increases the risk of pulmonary oedema.

- Cardiac valves can become progressively more calcified, particularly the aortic valve which can cause sclerotic disease and stenosis.

Respiratory:

- There is a decline in ventilatory response to hypoxia and hypercapnia.
- Loss of elasticity can increase lung compliance; however, chest wall compliance falls due to degenerative musculoskeletal changes, therefore total respiratory compliance often falls.
- Alveolar dead-space increases. Closing volume increases and may exceed functional residual capacity. Gas exchange is impaired across the alveolar membrane. The arterial oxygen tension falls with age.
- The upper airway is more likely to collapse from loss of elastance of the oropharynx, patients are at higher risk of sleep apnoea.
- Protective airway reflexes worsen with age, contributing to postoperative aspiration risk.
- Patients may be edentulous; this can make facemask ventilation difficult due to an inadequate seal; using an oropharyngeal airway may help.
- Postoperative respiratory complications are more common including pulmonary embolism and pneumonia. Patients are also more likely to have chronic lung disease.

Renal:

- Renal blood flow is decreased, the number of functioning glomeruli and overall renal mass falls which leads to a reduction in glomerular filtration rate. Creatinine clearance falls; however, creatinine may not itself rise due to a reduced creatinine production from a lower muscle mass.
- Renal homeostatic mechanisms worsen, with reduced ability to concentrate urine, reduced sensitivity to antidiuretic hormone (ADH) and a reduced response to renin-aldosterone. Thus, patients are more likely to develop abnormalities in fluid balance and hypo- and hypernatraemia are also more common.
- Reduced elimination of renally excreted drugs requires close attention to drug dosing.

Hepatic:

- Cell function is quite well preserved in healthy patients; however, there is a reduction in hepatic mass and blood flow. Hepatic excretion of drugs may therefore be reduced.

Central nervous system:

- Brain mass and neuronal mass decreases.
- Cerebrovascular disease is more prevalent.
- Cognitive impairment is more common. Cognitive decline affects around 20% of patients over 80 years old.
- There is an increased risk of postoperative delirium and postoperative cognitive dysfunction.
- Visual and hearing difficulty are more common which can impact on communication.
- Autonomic function is impaired, patients are more prone to postural hypotension. Delayed gastric emptying contributes to an increased risk of gastric aspiration.

- Older patients have a lower dose requirement for sedatives and opioids and are more likely to develop respiratory depression and reduced conscious level with these drugs.
- The thirst response is reduced, increasing the risk of fluid deficit.

Haematological/immunological:

- There is an increased risk of venous thromboembolism (VTE).
- Anaemia is more common, often unexplained and may be related to stem-cell ageing and erythropoietin resistance.
- Immunosenescence is a process of immune system deterioration with advancing age; the elderly are less able to respond to infection and heal wounds.

Metabolic and endocrine:

- The basal metabolic rate falls.
- Impaired thermoregulatory control, reduced subcutaneous fat and less effective shivering from a reduced muscle mass. Consequently, patients are more likely to become hypothermic.
- More likely to be malnourished.
- Endocrine disorders including diabetes and thyroid disorders are more likely. The elderly have a reduced ability to mount a stress response, have an abnormal glucose response and poor glycogen reserves.
- Arthritis is common, joint deformities can make regional anaesthesia difficult.
- Osteoporosis, fragile skin and fragile subcutaneous blood vessels contribute to patients being more susceptible to injury and bruising. Pressure sores are also more likely to occur; care must be taken when moving and positioning patients. Extravasation is more common when using intravenous fluids or drugs.

Musculoskeletal:

- Reduced muscle mass, ligamental weakness, osteoarthritic and osteoporotic skeletal changes.
- Soft tissue is more prone to injury when manoeuvred.
- Fragility fracture can occur and patients have a more challenging rehabilitation process after surgery.
- The range of motion of the cervical spine can be limited and this may make intubation more difficult.

What are the pharmacological considerations for the elderly?

There is altered drug distribution, metabolism and elimination. Impaired hepatic and renal function reduce drug excretion through these routes. There is a decrease in total body water which decreases the volume of distribution of water-soluble drugs, reducing the dose requirements. Fat percentage is higher, this increases the volume of distribution of lipophilic drugs which may prolong clearance.

Nearly all opioids and sedative-hypnotic agents have an age-related increase in elimination half-life. Clinically if using these drugs then lower drug doses and/or an increased dosing interval may be needed.

Reduced cardiac output delays onset of intravenous anaesthesia due to increased arm-to-brain circulation time.

There is a decrease in plasma albumin, which can increase free drug fraction.

Minimum alveolar concentration (MAC) decreases with age; age-adjusted MAC can be used.

There is increased sensitivity to central nervous system depressant drugs.

Despite a reduction in muscle mass, the effective dose of neuromuscular blocking agents is virtually unchanged. Time to onset of blockade may be slightly prolonged, partly due to a reduced cardiac output. Hepatic and renal impairment prolongs the effect of blockade in the agents that significantly rely on these organs for metabolism and elimination; this can be a factor to consider when choosing which neuromuscular blocking drug to use.

Polypharmacy with the use of multiple medications increases the possibility of interactions with drugs used during anaesthesia and in the perioperative period.

What are the preoperative, intraoperative and postoperative considerations in the older patient?

Preoperative:

A systematic review of the patient's medical, surgical and anaesthetic history should be taken and consider how their comorbidities impact on their daily life. History taking may be more difficult in this population and a collateral history from family members/carers can help in this context.

Social and functional history is valuable to understand a patient's level of physical ability and their dependence on others for activities of daily living. This helps identify patients who are higher risk. Exercise tolerance is an indicator of respiratory and cardiovascular reserve; however, it can often be limited by joint pain. Alcohol and smoking history should be taken.

Mental state should be assessed; cognitive impairment and delirium are more common. If it is thought that a patient may lack capacity, then they should be assessed for capacity in accordance with the Mental Capacity Act. Patients with cognitive impairment including dementia may still have full decision-making capacity and where possible those with dementia should be helped and encouraged to make cognitively demanding decisions. Capacity may also fluctuate; it may be possible to consent during a lucid period.

Investigations performed preoperatively will depend upon patient comorbidities and the extent of their surgery. If a patient has suffered a fall, the underlying medical cause should be investigated.

Pre-optimisation can reduce complications associated with disease; this includes perioperative management of comorbidities. This may involve a multidisciplinary team. However, the time taken to pre-optimize needs to be balanced against the risks of delaying surgery. This will depend on the individual patient and the surgical procedure being performed. For emergency surgery, such as emergency laparotomy or hip fracture surgery, optimisation and surgery are often done simultaneously rather than consecutively.

Older patients are more prone to ischaemia and aims should be to reduce oxygen uptake through good analgesia, thermoregulation and disease management, as well as optimising oxygen delivery, avoiding hypotension, treating severe anaemia and reviewing medications. Nutritional assessment can identify those who are nutritionally

deficient, management of which can improve healing and recovery. Prolonged preoperative fasting should be avoided where possible.

Intraoperative:

Choice of anaesthesia (regional or general) is dependent on the individual patient and neither is superior overall, it is the care taken during anaesthesia rather than the anaesthetic mode itself which is most important.

Additional monitoring should be considered. Intra-arterial blood pressure monitoring allows quick recognition of changing blood pressure, as well as facilitating the testing of arterial blood gases. Older patients usually require a lower dose of hypnotic agent for induction and are more sensitive to overdose from anaesthetic agents with myocardial depression, hypotension and delayed recovery. Depth of anaesthesia monitoring such as bispectral index (BIS) or entropy monitors can help guide depth of anaesthesia and sedation. Cerebral oxygen saturation monitoring and early intervention may reduce the prevalence of postoperative cognitive dysfunction, although further research is needed in this area. Cardiac output monitoring can be used to help guide fluid, vasopressor and inotropic therapy.

Perioperative hypothermia is common in the elderly and associated with delirium, cardiac complications, poor wound healing and prolonged hospital admission. Temperature can be maintained with warming devices such as warmed IV fluids, forced-air warmers and other body warming devices, as well as elevation of ambient temperature.

Fluid and electrolyte management is more challenging in the older patient; they have a reduced ability for homeostatic compensation following blood and fluid loss, but also following the administration of intravenous fluids.

Careful positioning is vital; it must consider the patient's musculoskeletal condition including arthritic joints as well as deformities. There is a higher risk of preventable peripheral nerve injury during prolonged surgery and at-risk sites should be appropriately padded and assessed throughout surgery. Elderly skin is friable and care needs to be taken when transferring the patient as well as removing adherent items from the skin. Reduced muscle mass, skin depth and vascularity mean older patients are at higher risk of preventable pressure sores, particularly over bony protuberances. Pressure sores can be complicated by infection and pain and contribute to prolonged recovery and hospital stay.

Postoperative:

It is recommended that anaesthetists routinely risk assess older patients towards the end of surgery with regard to the level of postoperative care they require, for example whether the patient requires enhanced care, high dependency care or intensive care.

The principles of good perioperative management extend into the postoperative period, including analgesia, maintenance of normothermia, fluid management, pressure care, nutrition, VTE prophylaxis and glucose control. Pain in the older patient contributes to morbidity including delirium, cardiovascular and respiratory complications, reduced mobilisation and prolonged length of stay. Assessment and management of postoperative pain in the elderly can be more challenging in those with cognitive impairment; older people may also be less likely to report pain. The use of perioperative analgesia protocols can improve patient satisfaction, but require individualisation to consider frailty, chronic pain, hepatic and renal function, concurrent medications and

cognitive impairment. Analgesia does not only include medications and regional techniques, but also attention to pressure care, positioning, postural support and warming.

Postoperative delirium and postoperative cognitive dysfunction are more common in the elderly. Identification and risk reduction should continue into the postoperative period. Delirium assessment tools include Diagnostic and Statistical Manual of Mental Disorders (DSM) and the Confusion Assessment Method (short CAM or CAM-ICU).

What is frailty?

A state of increased vulnerability to poor resolution of homeostasis after a stressor event.

Do you know any assessment tools for frailty?

Tools include the Edmonton Frail Scale (EFS) and the Clinical Frailty Scale (CFS).

The EFS is a 17-point scale, which is quick to perform. It involves asking a patient to draw numbers on a clock face, as well as questions about their general health, functional independence, social support, medications, nutrition, mood and continence. The 'get up and go test' which is part of the test may have value in preoperative anaesthetic clinics but is not applicable in most emergency situations.

The CFS is scored on a scale from 1 (very fit) to 9 (terminally ill). It considers a patient's level of physical activity, disease symptoms and dependence on others for daily help. Each stage has a descriptor of the level of fitness or frailty, accompanied by a visual representation.

What medications are used for dementia and how do these impact on anaesthesia?

Specific drug treatment for dementia will often involve the use of acetylcholinesterase inhibitors. In Alzheimer's disease, it is suggested that cognitive impairment is linked to a decrease in central cholinergic neurones. These drugs can prolong the effect of depolarising neuromuscular blockers and decrease or reverse the effect of non-depolarising neuromuscular blockers.

Decisions on whether to pause acetylcholinesterase inhibitors prior to elective surgery should be made on an individual basis taken in discussion with the patient and their relatives/carers where appropriate. Considerations include the likelihood of requiring neuromuscular blockade during surgery as well as the specific drug and its half-life. Rivastigmine and galantamine have short half-lives and can be discontinued the day before surgery. Donepezil, however, has a long half-life with a washout period of 2–3 weeks, discontinuing for this time period risks irreversible worsening of cognitive function. It may be more appropriate to continue donepezil and manage muscle relaxation accordingly with the use of neuromuscular monitoring to aid appropriate drug dosing.

Neostigmine may be ineffective due to pre-existing levels of cholinergic inhibition, or may even prolong neuromuscular blockade via a phase II block with succinylcholine. Alternative options include using short-acting agents that undergo spontaneous degradation such as atracurium, or using sugammadex to reverse rocuronium or vecuronium. If rapid sequence induction is indicated, rocuronium and reversal with sugammadex is probably the best option as it avoids prolonged blockade from succinylcholine and allows predictable reversal.

Memantine is an N-methyl-D-aspartate (NMDA) receptor antagonist that can be used as monotherapy in patients who are intolerant to or have a contraindication to acetyl cholinesterase inhibitors, or used as an additional therapy in those with moderate to severe dementia. It can enhance the central nervous system toxicity of ketamine and may enhance the side effects of dopaminergic or anticholinergic drugs.

Ginkgo biloba is an over-the-counter herbal remedy that may be taken by people with cognitive impairment or dementia. It increases the risk of bleeding due to interference with platelet function. It is recommended to discontinue it 2 weeks prior to surgery.

Patients may also be taking medications used to help non-cognitive symptoms. Selective serotonin reuptake inhibitors may be used for depression, there is a risk of potentiating serotonin syndrome for example when using drugs such as fentanyl, tramadol and ondansetron. Benzodiazepines and atypical antipsychotic agents may be used for symptoms of agitation, fear and hallucinations, these drugs can potentiate the effect of anaesthetic agents including neuro and cardio-depressant effects.

What is postoperative cognitive dysfunction (POCD)?

POCD can be described as a postoperative decline in cognitive function which persists after a patient has recovered from the acute impact of surgery. Mostly it resolves over time but can potentially last from months to years. It is not yet a formal psychiatric diagnosis and is not yet defined by the DSM. Lack of international consensus criteria for diagnosis contributes to the wide variation in quoted incidence between studies. Detection is usually delayed until at least 7 days after surgery because before this time there are multiple transient causes of cognition change that cannot be attributed to POCD (such as anaesthesia emergence, alterations to sleep, pain, polypharmacy, surgical complications and nutritional deficits). POCD can impair any of the cognitive domains including learning, memory, language, visual perception, motor skills, social function, attention and executive function. Symptoms can include difficulty with higher mental tasks such as concentrating on a story or film, making lists, multitasking, managing money and problem solving. It can be subtle and may not be noticed until weeks or months after an operation, where families or carers may notice that people find tasks difficult which they easily coped with before. Detection and testing are best done with a series of neuropsychometric tests covering the cognitive domains mentioned, with comparison to a preoperative baseline.

Do you know any risk factors for POCD?

POCD can occur in any age, but is more common in the elderly. Other risk factors include lower educational status, previous stroke, major surgery or reoperation during the same admission, longer duration of anaesthesia, postoperative infection and respiratory complications. Current research has not demonstrated a significant difference in the incidence of POCD after regional or general anaesthesia.

Further Reading

- Alcorn S, Foo I. Perioperative management of patients with dementia. *British Journal of Anaesthesia*. 2017; 17: 94–98.
- Allman K, Wilson I. 2016. *Oxford Handbook of Anaesthesia*. 4th ed. Oxford University Press.

- Association of Anaesthetists of Great Britain and Ireland. Peri-operative care of the elderly 2014. *Anaesthesia*. 2014; 69: 81–98.
- Brodier EA, Cibelli M. Postoperative cognitive dysfunction in clinical practice. *British Journal of Anaesthesia*. 2021; 21: 75–82.

- Cibelli M. Becoming confused after an operation. *Royal College of Anaesthetists*. 2017. www.rcoa.ac.uk/sites/default/files/documents/2019-07/07-Confused2017.pdf.
- Evers A, Maze M, Kharasch E. 2011. *Anesthetic Pharmacology. Basic Principles and Clinical Practice*. 2nd ed. Cambridge University Press.
- Griffiths R, Mehta M. Frailty and anaesthesia: What we need to know. *Continuing Education in Anaesthesia Critical Care and Pain*. 2014; 4: 273–277.
- Hacking R, O'Connor D. Anatomy and physiology of ageing. *Anaesthesia Tutorial of the Week* 2008. <https://resources.wfsahq.org/atotw/anatomy-and-physiology-of-ageing/>.
- Hollister N. Anaesthesia in the elderly. *Anaesthesia Tutorial of the Week* 2006. <https://resources.wfsahq.org/atotw/anaesthesia-in-the-elderly/>
- Murray D, Dodds C. Perioperative care of the elderly. *Continuing Education in Anaesthesia Critical Care and Pain* 2004; 4: 193–196.
- Pang CL, Gooneratne M, Partridge JSL. Preoperative assessment of the older patient. *British Journal of Anaesthesia*. 2021; 21: 314–320.
- Tan A, Amoako D. Postoperative cognitive dysfunction after cardiac surgery. *British Journal of Anaesthesia*. 2013; 3: 218–223.
- Yentis S, Hirsch N, Ip J. 2019. *Anaesthesia, Intensive Care and Perioperative Medicine A-Z*. 6th ed. Elsevier.

2.11.1 Abdominal Aortic Aneurysm Repair – Jessie R Welbourne and Kate Palmer

The anaesthetic management of abdominal aortic aneurysms (AAAs) is a big topic which can be evaluated in the CRQ and SOE. It is important to understand the effects of aortic cross-clamping and the management intra- and postoperatively of elective and emergency repairs.

What is an AAA?

An AAA is a permanent dilatation in the wall of the aorta > 30 mm in diameter. They develop due to loss of collagen and elastin fibres combined with poor quality cross-links, chronic inflammation of the vessel wall or loss of smooth muscle cells. The population incidence is 4.9–9.9% and most commonly occur in men aged > 65 years.

Atherosclerosis is the commonest cause but AAAs also occur in patients with connective tissues disorders such as Marfan's syndrome, arteritis e.g. Takayasu's disease, infections such as TB, syphilis and salmonella, post-trauma and inflammatory conditions.

Small aneurysms are usually asymptomatic but can expand over time and can rupture. Rupture risk increases in a non-linear fashion becoming significant once over 5 cm. There is an ultrasound screening programme for men aged 65 years in the UK or those with risk factors, with a detection specificity and sensitivity of nearly 100%.

What are the risk factors for developing an AAA?

A positive family history is the strongest predictor of an AAA developing. Men are six times more likely to develop AAAs, although women who develop them have a higher mortality rate and risk of rupture. Smokers are four times more likely than non-smokers to have an aneurysm that grows faster.

Other risks include hypercholesterolaemia, hypertension, diabetes, Caucasian background, coronary artery and peripheral vascular disease.

What treatment options are available?

These can be divided into medical and surgical management.

Medical management aims to prevent further aneurysm expansion and is directed towards modifiable risk factors (e.g. smoking) and coexisting comorbidities control. In animal models indomethacin, tetracyclines and statins have been beneficial but not in humans.

Surgical or endovascular repair is the definitive treatment option. The aneurysm is replaced with a synthetic graft. Elective surgery has much lower morbidity and mortality than emergency surgery for ruptured aneurysms. Mortality for elective repair is 7% but 36–50% for an emergency repair. Patients who survive the initial procedure are still at high risk of complications.

Elective surgery is performed when the aneurysm is > 5.5 cm diameter, either as an open repair or by endovascular stenting. The choice of technique depends on aneurysm morphology and the patient's age and comorbidities and is made by an MDT including interventional radiologists and vascular surgeons.

Conservative management of ruptured aneurysms is considered in patients unlikely to survive or who decline surgery.

Do you know any scoring systems assessing risk?

General scoring systems such as APACHE II and POSSUM estimate risk but do not accurately predict individual outcome in ruptured AAA patients.

Goldmann and Detsky analysis both assess cardiac risk factors and provide a score equating to relative risk of a major adverse cardiac event in non-cardiac surgery.

The Hardman index (1996) gives a score from 0 to 5 with a point awarded for age > 76 , serum creatinine > 190 $\mu\text{mol/L}$, haemoglobin < 90 g/L, ischaemic changes on a 12-lead ECG and a history of loss of consciousness after arriving in hospital. A score of ≥ 2 equates to a predicted mortality of 80%.

The Glasgow Aneurysm Score (GAS) applies to elective and emergency repairs. It adds age in years to other variables to give a total score. Variables include shock, myocardial disease, cerebrovascular disease and renal disease. A GAS score of 84 equates to a 65% mortality.

Scores should only be used to support clinical decision-making although they can also be used to compare different centre's outcomes.

A patient's exercise tolerance should be recorded. The ability to climb one flight of stairs without symptoms indicates an exercise capacity of ≥ 4 metabolic equivalents (METs). METs can give an indication of functional capacity. Patients achieving ≥ 4 METs have a better outcome. This is formally measured by VO_2 max during cardiopulmonary exercise testing. An anaerobic threshold/ VO_2 max ≤ 11 ml/kg/min predicts high perioperative risk.

How may a ruptured AAA present?

When an AAA ruptures, patients usually have abdominal or back pain with a pulsatile abdominal mass. They may be profoundly shocked. Differential diagnoses include bowel ischaemia, perforated viscus or pancreatitis.

Free intraperitoneal rupture almost always results in cardiovascular collapse and death. Retroperitoneal rupture allows blood to tamponade, limiting further haemorrhage, and has a better prognosis.

What are the important points when anaesthetising patients for an emergency AAA repair?

It is important to provide a structured answer. This is a surgical emergency and you are likely to have an unstable patient with comorbidities. Informing the examiner of this shows you understand the key issues.

A patient with a ruptured AAA is a surgical emergency requiring immediate repair in theatre. The management can be divided up into pre-, intra- and postoperative phases.

Preoperatively the patient may be profoundly shocked requiring resuscitation. They are likely to be elderly with several comorbidities. My aims are to stabilise the patient without wasting time. Theatres should be informed and transfer made with minimal delay. I would need the help of a senior anaesthetist and assistant.

I would quickly assess the patient using an ABC approach. I would administer high-flow oxygen and insert two wide-bore peripheral cannulae. I would take bloods for FBC, U&Es, clotting, cross-match and a venous gas. The blood bank should be alerted via the major haemorrhage protocol and discussion should take place with them around the need for large volume blood product replacement. This would include ongoing need for packed red cells, fresh frozen plasma, cryoprecipitate and platelets.

The patient should be judiciously resuscitated to enable them to get to theatre. Over resuscitation may dilute clotting factors and lead to worsening bleeding. It is important to avoid hypertension, coughing or straining as this may dislodge an arterial thrombus. The patient may require analgesia such as IV morphine or fentanyl targeted to effect.

A focussed anaesthetic assessment should be made including an airway assessment, past medical history and allergies.

What other equipment would you need?

Ideally there should be immediate access to cell salvage, a rapid infuser and point-of-care testing e.g. a HaemoCue and TEG/ROTEM. You may also consider cardiac output monitoring.

Cell salvage should be used from the start to maximise the amount of autologous blood collected. Cell salvage blood only contains red cells, not clotting factors or platelets, and cannot be transfused under pressure as this can precipitate haemolysis. Other blood products may still be needed.

A rapid infuser allows warmed fluid to be infused under pressure from an automated chamber. At the same time a second bag for infusion can be prepared without interrupting administration. It allows for the highest flow rate of fluid administration. The main risks of using a rapid transfer include air embolus which may be high enough in volume to result in harm and the risk of over-transfusion resulting in polycythemia.

Assuming the theatre is ready, how are you going to anaesthetise this patient?

It is important to state confidently what you are going to do, giving details. You need to demonstrate you understand the challenges, have managed these types of cases and are competent and safe. There is no single 'right answer' but choose one technique and be prepared to explain your choice.

This is a surgical emergency and I would anaesthetise the patient in theatre, on the table and with two arm boards, draped, with the surgeons scrubbed and ready. My goal in anaesthetising the patient is to maintain a cardiovascular stable anaesthetic and normothermia.

I would have a second experienced anaesthetist with me and emergency drugs prepared including fluids and inotropes. I would induce anaesthesia using a rapid sequence induction with 1:1:1 ketamine:fentanyl:rocuronium and cricoid pressure. Ideally I would have invasive arterial monitoring, a central venous catheter and urinary

catheter already inserted but would not delay surgery establishing these. I would like, ideally cross-matched, blood available and checked.

After induction I would maintain anaesthesia with a volatile agent, air/oxygen mixture, opiate analgesia and neuromuscular blockade.

I would monitor the patients' temperature and avoiding hypothermia, use a blood warmer, warming mattress and a forced-air warmer on the top half of the patient but not on their lower limbs as it may worsen ischaemic injury.

What are the physiological responses to aortic cross-clamping?

This is likely to be asked in any question about aneurysms and you need to clearly explain the physiology.

When the aorta is clamped there is a sudden increase in systemic vascular resistance and BP. This increased afterload raises the left ventricular end-diastolic BP leading to increased myocardial work and reduced coronary perfusion pressure. This may precipitate left ventricular ischaemia or failure. Increasing the depth of anaesthesia or using nitrates may attenuate this response.

What happens when the clamp is removed?

When the clamp is removed there is a sudden drop in afterload while metabolites from the ischaemic tissues re-enter the circulation. This can lead to lactic acidemia, tachycardia and a fall in BP. This may result in myocardial ischaemia and, if poorly managed, circulatory collapse. BP should be maintained with adequate fluid resuscitation before clamp removal in addition to administering vasoconstrictors or inotropes.

What is the importance of supra or infra-renal clamps?

The clamp position depends on the aneurysm location and how it relates to the renal arteries. If the clamp is supra-renal the kidneys blood supply is compromised increasing the chance of an ischaemic injury and renal failure. Clamp time should be minimised.

The anaesthetist can help by maintaining normo-tension and normo-volaemia aiming to reduce the risk of secondary injury. There is no evidence diuretics improve outcomes. Nephrotoxic agents e.g. NSAIDs, should be avoided.

What are the postoperative care aims?

Patients should be cared for on ICU. The aim is to restore normal homeostasis, normothermia, normal clotting and ventilate until a metabolic acidosis has resolved. Patients may require cardiovascular and renal support. Fluid management should include maintenance and boluses to correct hypotension or inadequate urine output. There is a significant risk of acute renal failure requiring renal replacement therapy (RRT). Analgesia should be administered including the use of local anaesthetic infusions e.g. via rectus sheath catheters.

What complications can occur?

Intraoperatively bleeding can occur at the anastomosis site after clamp removal and, if severe, may require reapplication. The anaesthetist can help by achieving normothermia and normal clotting.

Perioperative complications:

- Myocardial ischaemia and infarction due to changes in volume status, perfusion pressure and SVR
- Thromboembolism and organ ischaemia e.g. cerebral infarction, renal ischaemia and resultant acute kidney injury
- Hypoperfusion to the spinal cord – due to hypoperfusion and reduced flow via the anterior spinal artery or occlusion due to clamping. This may result in acute spinal cord injury.

Postoperative complications:

- Acute renal failure requiring RRT
- Infection e.g. respiratory or via indwelling catheters
- Abdominal compartment syndrome – advise monitoring the pressure via a urinary catheter probe
- Paralytic ileus – due to bowel ischaemia and may warrant TPN.

How can patients be optimised before elective repair?

Management should be directed to modifiable risk factors. Patients should stop smoking and, if diabetic, maintain tight blood sugar control. Patients may need to see specialist physicians e.g. cardiology if there's evidence of coronary artery disease. Statins and potentially beta-blockers should be prescribed. Low-dose aspirin should be started when an AAA is detected and continued indefinitely.

Tell me about endovascular aneurysm repair (EVAR)

EVAR is less invasive and avoids a laparotomy. Initially there is a lower mortality rate but this changes with time and survival post-procedure.

EVAR is usually performed in the radiology department via a femoral arterial line. Some patients can tolerate the procedure without general anaesthesia (GA) but many require converting to GA due to severe back, abdominal or ischaemic leg pain. They must be able to tolerate lying flat for several hours.

Further Reading

Al-Hashimi M, Thompson J. Anaesthesia for elective open abdominal aortic aneurysm repair. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2013; 13(6): 208–212.

Leoard A, Thompson J. Anaesthesia for ruptured abdominal aortic aneurysm. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2008; 8(1): 11–15.

2.11.2 Anaesthesia for Carotid Endarterectomy – Will H Spencer, Emily K Johnson and Jessie Welbourne

Questions about carotid endarterectomy feature in both the written and structured oral examinations (SOEs). Since the GALA trial 14 years ago, the procedure has been offered with either local anaesthesia or general anaesthesia.

What is a carotid endarterectomy?

Carotid endarterectomy (CEA) is a vascular surgical procedure that aims to reduce the risk of embolic stroke by treating severe carotid artery stenosis. It involves the surgical removal of atheromatous plaque from the lining of the internal carotid artery. The diseased intima and a portion of the media may be removed.

What are the indications for surgery?

Carotid endarterectomy is a prophylactic operation that aims to reduce the future risk of stroke. As such, careful evaluation of patient-specific risks versus benefits is required.

Two large randomised controlled trials inform which patients should be referred for carotid endarterectomy:

1. The NASCET trial (1998) demonstrated that for every six patients treated, one major stroke was prevented at 2 years for symptomatic patients with 70–99% stenosis.
2. The European Carotid Surgery Trial (ECST) (1998) showed NNT of 9 at 3 years in those with 80–99% stenosis. CEA did not appear to benefit patients with <80% stenosis.

NICE guidelines advise that everyone with a transient ischaemic attack or non-disabling stroke, who after specialist assessment is considered a candidate for carotid endarterectomy, should be referred for urgent carotid imaging. Those with 50–99% according to the NASCET criteria or 70–99% according to ECST criteria warrant urgent referral for carotid endarterectomy. Ideally, the operation should be undertaken within 14 days of symptom onset.

What about asymptomatic patients?

The ACST-1 study (2003) and a recent meta-analysis by Halliday et al. (2021) suggest that successful CEA halves the long-term risk of stroke in asymptomatic patients with severe carotid stenosis. The decision to perform CEA needs to be weighed up against individualised perioperative risk, the risk of future stroke, and life expectancy.

What are the risks associated with carotid endarterectomy?

CEA is a high-risk procedure often performed in a high-risk patient group. Risks can be divided into patient-specific and procedural risks.

Patient-specific risks: these patients often carry multiple characteristics that increase their perioperative risk. For instance, they are more likely to be elderly, smokers, hypertensives, with poorly controlled diabetes. They are more likely to have ischaemic heart disease, cerebrovascular disease, renovascular disease, and smoking-related lung disease. Most of these patients will have had a recent TIA or stroke, which significantly increases the risk of subsequent stroke. Furthermore, blood pressure is often labile following a stroke, and can be a challenge to manage.

Procedural risks: CEA is a high-risk procedure carrying a 30-day mortality of approximately 1% and a 30-day perioperative stroke rate of 2–3% (Reddy 2019). It also carries a risk of perioperative myocardial infarction. Haemorrhage can be sudden and catastrophic. Localised haematoma and oedema can cause tracheal compression and airway compromise. Recurrent laryngeal and hypoglossal nerve damage can occur

during surgery. Furthermore, there is approximately a 1% risk of cerebral hyperperfusion syndrome and potential haemorrhagic stroke. Performing the procedure awake may allow detection of this and appropriate surgical intervention to avoid stroke.

Can you tell me more about cerebral hyperperfusion syndrome?

Cerebral hyperperfusion syndrome is a potentially life-threatening condition characterised by marked hypertension in the postoperative period. It can present with headaches, visual change, neurological deficits, and seizures. It occurs in approximately 1% of patients undergoing CEA and tends to present 2–7 days post-procedure. It is likely caused by ischaemia – reperfusion injury and subsequent loss of autoregulation to the affected side of the brain. If untreated, it can progress to cerebral oedema, haemorrhagic stroke, and death. It is imperative that blood pressure is monitored closely, and hypertension treated expediently, in the postoperative period.

Do you know of any risk factors that increase the risk of perioperative morbidity and mortality?

Increased risk is associated with:

Patient factors:

- Increasing age (>75)
- Female sex
- Recent stroke or TIA
- Acute stroke as indication
- Hypertension
- Diabetes on insulin
- Coronary artery disease
- Severe pulmonary dysfunction

Surgical/anatomical risk factors:

- Previous radiotherapy
- Previous neck dissection
- Previous ipsilateral CEA
- Deep carotid plaque ulcer
- High carotid bifurcation
- Contralateral carotid stenosis

What anaesthetic options are available?

CEA can be performed under local or general anaesthesia. Local infiltration can be performed by administering large volumes of dilute local anaesthetic to tissue planes from the incision to the carotid sheath. Regional anaesthesia techniques include superficial, intermediate, and deep cervical plexus blocks. These techniques can be supplemented by blocking the mandibular branch of the trigeminal nerve via the intraoral approach. Cervical epidural has been described but is generally avoided in the UK due to associated risks.

What is the evidence relating to the use of general anaesthesia or local anaesthesia for carotid endarterectomy?

The GALA (general anaesthesia versus local anaesthesia) trial (2008) is a randomised controlled trial that compared general with local anaesthesia for carotid endarterectomy. It concluded there was no statistically significant difference in stroke, myocardial infarction, or death between the two groups. These findings have been supported by subsequent Cochrane reviews (Rerkasem et al., 2008 and Vaniyapong et al., 2013). More recent studies suggest a potential advantage in regional anaesthesia in terms of patient outcome, cost-effectiveness, and length of stay. Ultimately, the anaesthetist and surgeon should consult with the patient and decide on the most suitable anaesthetic technique on an individualised basis.

Can you tell me a little more about the nerves you would need to block to perform a carotid endarterectomy under local anaesthetic?

The cervical plexus originates from the ventral rami of C1 to C4. The deep branches are solely motor and supply the deep muscles of the neck. The superficial nerves supply sensation over the anterolateral aspect of the neck. The superficial cervical plexus is located at the lateral border of sternocleidomastoid and comprises four nerves:

1. Lesser occipital nerve
2. Greater auricular nerve
3. Transverse cervical nerve
4. Supraclavicular nerve

Surgical retraction of submandibular tissues can cause pain in the mandibular region of the trigeminal nerve, particularly in those with short necks or a high carotid bifurcation. Intraoral mandibular nerve blocks may help alleviate this. The carotid sheath also has cranial nerve innervation requiring local anaesthetic infiltration by the surgeon.

Describe the technique for performing a superficial cervical plexus block?

When asked to describe any nerve block do not forget to mention the preparations you would make. If the examiner is not interested in this, they can stop you, but it is important to demonstrate you are safe and thoughtful. If you can, quickly sketch a diagram to support your answer (Figure 2.11.2.1).

Prior to performing any nerve block I would take a full history and examine the patient. Once establishing their suitability and ruling out any contraindications I would explain the procedure and risks and consent the patient. I would prepare my equipment and monitoring and have a trained assistant and emergency equipment to hand.

Firstly, I would gain intravenous access, attach monitoring, and administer oxygen, giving sedation if indicated.

For the landmark technique, I would position the patient supine with some head-up tilt and the head turned slightly away from the side of the block. The landmark is the midpoint of the lateral border of sternocleidomastoid muscle (SCM). Using a sterile technique, I would insert a 21-gauge needle along the posterior border of SCM both cephalad and caudad, puncturing the first fascial layer, aspirating and injecting a total of 10 ml of 0.25% bupivacaine. I would expect a sausage shape to form along the lateral border of SCM.

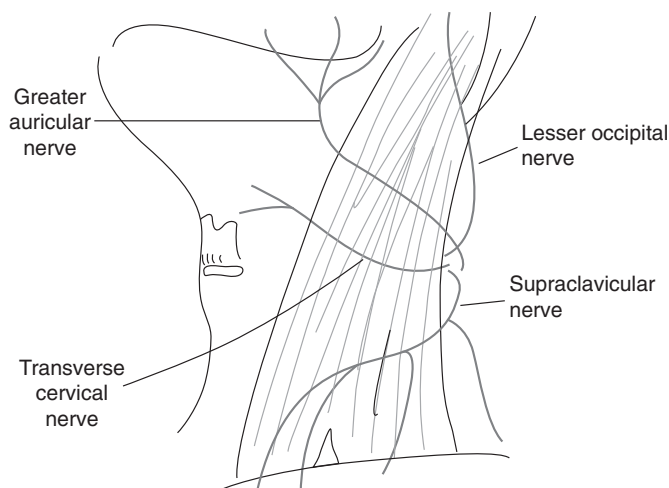


Figure 2.11.2.1 Superficial cervical plexus.

For an ultrasound-guided block, I would position the patient in the lateral position with their head comfortably supported. I would then place a high-resolution linear probe transversely over the midpoint of the lateral border of SCM and use dynamic scanning to determine the underlying structures. The superficial cervical plexus should be visualised as a bundle of hypoechoic nodules deep to SCM and superficial to the interscalene groove. Using aseptic technique, I would introduce a 50 mm nerve block needle and guide it to a position adjacent to the plexus. After negative aspiration, I would slowly inject 10 ml of 0.25% bupivacaine and visualise the spread of local anaesthetic under ultrasound.

Describe the technique for performing a deep cervical plexus block.

I would prepare the patient as previously mentioned and position the patient supine with some head-up tilt with the head turned away from the side of the block (Figure 2.11.2.2). I would palpate the cervical transverse process at the level of C4, behind the lateral border of SCM. Using aseptic technique, I would anaesthetise the skin with local anaesthetic before introducing a 50 mm nerve-block needle. I would direct the needle in a slight posterior and caudal direction until the C4 transverse process is encountered. After careful aspiration I would then inject 8–10 ml of 0.25% bupivacaine. This block can also be performed under ultrasound by directing a needle onto the C4 transverse process.

What are the risks associated with a deep cervical plexus block?

When asked the risks of any nerve block you can classify them into the generic risks of local anaesthetic injection and those specific to a particular block.

The risks can be classified into those due to local anaesthetic injection, such as local anaesthetic toxicity, anaphylaxis and nerve damage, and those specific to deep cervical plexus blocks. These include:

- Intravascular injection (specifically carotid or vertebral arteries)
- Nerve palsies: phrenic nerve causing diaphragmatic weakness (never perform this block bilaterally), recurrent laryngeal nerve damage causing vocal cord paralysis, sympathetic block causing a Horner's syndrome

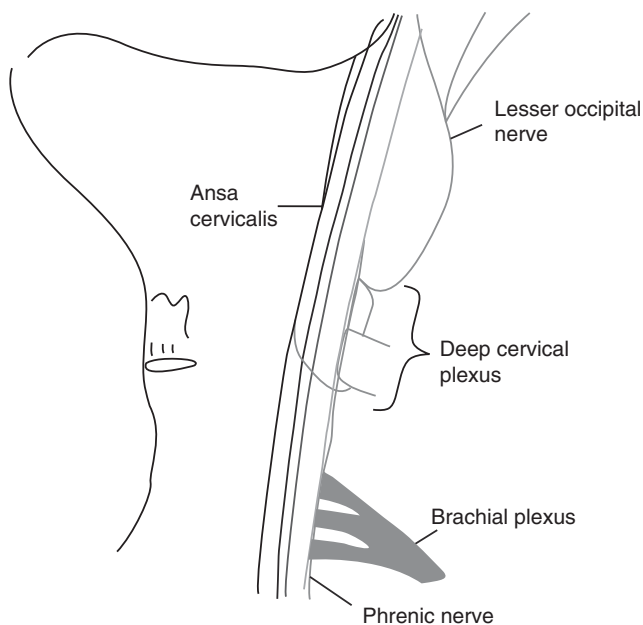


Figure 2.11.2.2 Deep cervical plexus.

- Epidural spread
- Intrathecal injection
- Spinal cord damage.

What is meant by an intermediate cervical plexus block?

This is when local anaesthetic is infiltrated between the investing layer of the deep cervical fascia and the prevertebral fascia. It is associated with lower frequency of complications when compared to a deep cervical plexus block.

What are the advantages and disadvantages of using a local anaesthetic technique?

Local anaesthetic techniques allow continuous monitoring of higher cerebral, motor, and sensory functions without the need for additional specialist equipment. This is done by maintaining verbal contact with the patient throughout the operation. This can allow the surgeons to be more selective about when to insert a shunt. Local anaesthesia also avoids the risk of general anaesthesia in what is often a high-risk group. Cerebral autoregulation is better preserved under local anaesthesia compared with general anaesthesia.

Disadvantages include potential poor compliance during a potentially long operation, patient discomfort, and movement of the surgical field. Anxiety-related tachycardia and hypertension may make haemostasis more challenging and may precipitate myocardial ischaemia. Patient anxiety may also lead to an increased cerebral metabolic rate and, therefore, increased oxygen demand during a time when supply may be reduced. If the need arises to convert to a general anaesthetic, this can present practical issues in terms

of airway access and disrupting the operation. The complications of a deep cervical block are outlined above.

If you were to choose a regional technique, which would you choose?

Feel free to express your own opinions when asked a question about personal choice, however, be prepared to justify your choices.

I would personally choose the technique most familiar to me: a combination of a superficial cervical plexus block with local infiltration at the incision site. There is a randomised controlled trial comparing deep and superficial block with superficial block alone. It demonstrated the techniques were comparable in the amount of supplemental local anaesthetic given by the surgeon. Anatomically, a superficial block alone would not be expected to block all the relevant nerves; however, dye studies suggest that as long as the injection is below the investing fascia, local anaesthetic may spread from superficial to deep structures. A superficial block alone avoids the hazards of the deep cervical plexus block. I would supplement this with judiciously titrated TCI remifentanyl.

What are the principles of general anaesthesia for carotid endarterectomy?

The key principles for endarterectomy under general anaesthesia are adequate airway protection, ventilatory control, and cardiovascular stability in order to maintain an adequate cerebral perfusion pressure and oxygenation. General anaesthesia also has the potential benefit of reducing the cerebral metabolic rate for oxygen (CMRO₂) at a time when blood flow may be impaired. In an awake case, if the key risk of cerebral perfusion compromise were to arise intraoperatively, this can be rapidly assessed and acted on because the patient is able to demonstrate a change in function. For cases under general anaesthesia, this functional assessment is not possible and so several surrogates of cerebral perfusion assessment are used, including EEG monitoring and carotid stump pressures. The principles of anaesthesia are to optimise cerebral perfusion and provide a cardiovascularly stable anaesthetic.

Airway protection with an endotracheal tube is advised as access to the airway can be compromised during surgery. Also, a laryngeal mask may alter the anatomy of the surgical site. Adequate ventilatory control is essential to ensure the patient is well oxygenated and arterial CO₂ tensions can be manipulated. Normocarbica should be maintained to avoid undesirable effects of hyper- or hypocapnia on cerebral blood flow. Haemodynamic stability is essential for maintaining an adequate cerebral perfusion pressure. Intra-arterial blood pressure monitoring should be established pre-induction due to the likelihood of coexisting cardiovascular disease and instability during induction. Blood pressure should be maintained within 20% of baseline values and can be augmented with vasopressors, particularly if there are any signs of cerebral ischaemia following cross-clamping. I would take care to avoid intraoperative hypotension and to ensure blood pressure at least a baseline value when the artery is being closed. This can help minimise the risk of postoperative bleeding. I will ensure the patient receives sufficient multimodal analgesia to reduce the risk of postoperative hypertension, myocardial ischaemia, and bleeding. I would personally use a TIVA technique to facilitate control of blood pressure, smooth emergence, and reduced coughing on extubation.

How may cerebral perfusion be monitored during general anaesthesia?

Cerebral perfusion may be assessed using processed EEG monitoring, measuring jugular venous oxygen tension, transcranial Doppler (TCD) or carotid artery stump pressure. The EEG gives a crude measure of the trend in brain electrical activity and is largely unspecific and dependent on correct application of surface electrodes. A low jugular venous oxygen tension indicates high oxygen uptake by the brain and so lower blood flow assuming the oxygen consumption by the brain remains stable. Transcranial Doppler can be used to measure blood flow in the middle cerebral artery and, therefore, give an estimate of cerebral blood flow as well as an indication of particulate emboli. Carotid artery stump pressure measurement is a crude method to estimate adequacy of perfusion. A mean pulsatile pressure of more than 50mmHg is accepted as sufficient to proceed without a shunt.

How would you manage cerebral ischaemia upon carotid cross clamping?

The initial management would be to administer supplemental oxygen and increase the mean arterial pressure to 20% above baseline to support collateral perfusion. If signs of cerebral ischaemia persist, the surgeon may opt to insert a shunt immediately or release the cross-clamp while a shunt is prepared.

What are the potential issues with inserting a shunt?

Shunts are associated with potential complications such as air or plaque embolisation, intimal tears, carotid dissection, and late carotid re-stenosis. Even following shunt insertion, flow may be insufficient to meet cerebral oxygen requirements.

Where should these patients be looked after postoperatively?

Postoperative care should be provided on a high dependency unit (HDU) level 1 or a specialised area that can provide an appropriate level of monitoring and expertise. This is so that any complications arising from the procedure can be recognised and managed expeditiously. Life-threatening airway compromise can occur from bleeding, oedema, and/or vocal cord dysfunction. Hypertension is common following endarterectomy and must be managed promptly to prevent complications such as surgical site bleeding, cerebral hyperperfusion syndrome or intracranial haemorrhage. Neurological deficit occurs in up to 7% of patients and could potentially warrant airway support and the need for further investigation.

Do you know of alternative treatments for carotid artery stenosis?

Carotid artery stenting is a viable alternative to endarterectomy and can be particularly useful in high-risk surgical patients. The procedure is carried out under local anaesthesia using a percutaneous transfemoral approach. A recent Cochrane review (Muller et al., 2020) concluded that while the risk of periprocedural stroke is higher with stenting than with endarterectomy, the two treatments are similar in preventing recurrent stroke following a successful procedure.

Further Reading

- Barnett HJ, et al. Benefit of carotid endarterectomy in patients with symptomatic moderate or severe stenosis. *The New England Journal of Medicine*. 1998; 339(20): 1415–1425.
- ECST Writers. Randomised trial of endarterectomy for recently symptomatic carotid stenosis: Final results of the MRC European Carotid Surgery Trial (ECST). *The Lancet*. 1998; 351 (9113): 1379–1387.
- GALA Trial Collaborative Group. General anaesthesia versus local anaesthesia for carotid surgery (GALA): A multicentre, randomised controlled trial. *The Lancet* 2008; 372 (9656): 2132–2142.
- Halliday A et al. Carotid artery surgery to reduce long-term stroke rates: Individual patient data eta-analysis of the randomised trials in asymptomatic patients. *The Lancet*. 2021.
- Halliday A et al., 10-year stroke prevention after successful carotid endarterectomy for asymptomatic stenosis (ACST-1): a multicentre randomised trial. *The Lancet*. 2010; 376 (9746): 1074–1084.
- Hussain AS, Mullard A, Oppat WF, Nolan KD. Increased resource utilization and overall morbidity are associated with general versus regional anesthesia for carotid endarterectomy in data collected by the Michigan surgical quality collaborative. *Journal of Vascular Surgery*. 2017; 66: 802–809.
- Knappich C, Kuehn A, Tsantilas P, et al. Intraoperative completion studies, local anesthesia, and antiplatelet medication are associated with lower risk in carotid endarterectomy. *Stroke*. 2017; 48: 955–962.
- Ladak N, Thompson J. General or local anaesthesia for carotid endarterectomy? *Continuing Education in Anaesthesia Critical Care and Pain*. 2012; 12(2): 92–96.
- Liu J, Martinez-Wilson H, Neuman MD, et al. Outcome of carotid endarterectomy after regional anesthesia versus general anesthesia: A retrospective study using two independent databases. *Translational Perioperative and Pain Medicine*. 2014; 1: 14–21.
- Muller MD, Lyrer P, Brown MM, et al. Carotid artery stenting versus endarterectomy for treatment of carotid artery stenosis. *Cochrane Database of Systematic Reviews*. 2020.
- National Institute for Health and Clinical Excellence. Carotid artery stent placement for symptomatic extracranial carotid stenosis. www.nice.org.uk/guidance/ipg389.
- National Institute for Health and Clinical Excellence. Stroke: Diagnosis and initial management of acute stroke and transient ischaemic attack (TIA). www.nice.org.uk/guidance/ng128.
- Reddy RP, Karnati T, Massa RE et al. Association between perioperative stroke and 30-day mortality in carotid endarterectomy: A meta-analysis. 2019; *Clinical Neurology and Neurosurgery*. 181: 44–51.
- Rerkasem A, Orrapin S, Howard DPJ, Rerkasem K. Carotid endarterectomy for symptomatic carotid stenosis. *Cochrane Database of Systematic Reviews*. 2020.
- Siu A, Patel J, Prentice HA, et al. A cost analysis of regional versus general anesthesia for carotid endarterectomy. *Annals of Vascular Surgery*. 2017; 39: 189–194.
- Stoneham MD, Stamou D, Mason J. Regional anaesthesia for carotid endarterectomy. *British Journal of Anaesthesia*. 2015; 114 (3): 372–383
- Vaniyapong T, Chongreksut W, Rerkasem K. Local versus general anaesthesia for carotid endarterectomy. *Cochrane Database of Systematic Reviews*. 2013.
- Warlow CP. Symptomatic patients: the European Carotid Surgery Trial (ECST). *Journal des maladies vasculaires*. 1993; 18(3): 198–201.

2.11.3 Anaesthesia for Lower Limb Peripheral Arterial Surgery – Gary Thomas

Which patient risk factors are associated with the development of peripheral arterial disease (PAD)?

The risk factors can be classed into two groups, those that are modifiable and those that are not. Modifiable factors include diabetes mellitus, smoking, dyslipidaemias, hypertension and obesity. Factors that cannot be influenced are age, male gender and a genetic predisposition.

How might a patient with peripheral lower limb arterial insufficiency present?

A patient may have asymptomatic disease and during simple clinical examination the peripheral pulses may be reduced or absent. This can be assessed by palpation of the popliteal or dorsalis pedis arteries in the lower limbs or radial and ulnar arteries in the upper limbs.

The legs or feet may be ulcerated, exhibit differences in skin temperature, loss of muscle bulk and trophic skin changes.

If peripheral arterial insufficiency is suspected, an ankle brachial pressure index or ABPI can be determined.

Tell me how you would perform an ABPI?

This is performed by measuring the systolic blood pressure of the brachial arteries by using a blood pressure cuff and Doppler ultrasound probe. Similarly, a cuff is placed on the lower leg just proximal to the malleoli and both the dorsalis pedis and posterior tibial systolic pressure values recorded.

An ankle brachial index (ABI) is calculated in each leg. For example, the right ABI is the highest pressure in either artery in the foot divided by the highest in either brachial artery. A ratio below 0.9 is considered diagnostic of peripheral vascular disease. A value of less than 0.5 signifies severe arterial disease.

Otherwise, a patient may present acutely or chronically.

The symptoms of **chronic arterial insufficiency** of the lower limb include paraesthesia, cold extremities and intermittent claudication of the calf muscles or buttocks eventually leading to rest pain.

Acute lower limb ischaemia is associated with limb pain at rest and a pulseless, painful foot. The likely cause is a proximal vascular thrombus added to pre-existing mild to severe atherosclerotic lesions. There is usually little or no evidence of a collateral circulation.

The six 'Ps' of acute arterial occlusion are: pain, pallor, pulselessness, poikilothermia, paraesthesia and paralysis.

You are asked by the vascular surgical team to review a 74-year-old man with acute vascular insufficiency of the left lower leg. He has had a cold, white painful lower leg for approximately 2 hours. He is known to have ischaemic heart disease, atrial fibrillation and mild chronic obstructive pulmonary disease (COPD). He is scheduled for an emergency left femoral embolectomy under a general anaesthetic. His current

oral medication is ramipril 10 mg once daily, aspirin 75 mg once daily and simvastatin 40 mg once daily.

His pulse rate is 115 beats/min, blood pressure 140/90 mmHg, oxygen saturation on air is 94% and he is apyrexial. Auscultation of his chest is clear.

How will you proceed?

This clinical scenario is typical of an SOE, particularly a short case. A systematic and concise approach will send the right signals to the Examiners.

Often the opening question for the Examiner is, 'Give me a summary of this case?'

I would assess him on the ward with a view to asking the Surgical team to consider performing the procedure under local anaesthetic infiltration, with or without sedoanalgesia. I would be present for the procedure and be ready to administer a general anaesthetic or perform a central neuraxial block.

My assessment will focus on the severity of his ischaemic heart disease, COPD and whether the atrial fibrillation was of recent onset. His atrial fibrillation may require rate control but anxiety and pain may be contributing factors with his tachycardia.

I would review haematological, biochemical results and an admission 12-lead ECG. I would review his notes for previous ECGs and whether echocardiography had been performed in the past.

It also would be appropriate to assess his acid-base status with arterial blood gas measurement.

The key to managing this patient is communication with the Surgical team and it is highly likely that they would be happy to perform this procedure under local anaesthetic infiltration.

However, there may be factors that might preclude this being performed under LA infiltration. These might be an inability to lie flat because of cardiac or respiratory causes or confusion.

How can symptomatic peripheral vascular disease be managed?

It can be managed conservatively and surgically.

The aim is to improve the patient's quality of life by reducing the symptoms of ischaemia and secondarily, reduced cardiovascular risk, morbidity and death.

Simple lifestyle changes include smoking cessation and exercise, the latter, paradoxically increases claudication distance.

Pharmacological treatment with cilostazol is known to increase pain-free walking time and distances. It is thought to act by decreasing platelet aggregation, vasodilatation and improved lipid profile. The introduction of statins significantly improves claudication distance and reduces the potential for other cardiovascular events.

While the antithrombotic therapies aspirin and ADP-antagonists clopidogrel or ticlopidine do not improve symptoms, they also reduce cardiovascular complications associated with atherosclerosis.

Apart from clinical examination, what investigations might establish the severity of peripheral vascular disease?

The first-line imaging modality for all patients with peripheral arterial disease is duplex ultrasound: before revascularisation, contrast-enhanced magnetic resonance

angiography. If contrast-enhanced magnetic resonance angiography is contraindicated or not tolerated then computed tomography (CT) angiography should be considered. These radiological investigations will determine whether the anatomy of the arterial system is suitable for an endovascular intervention or open revascularisation.

What is duplex ultrasound?

This refers to the use of ultrasound to visualise blood flow and the structure of blood vessels. Here two modes are used: Doppler and B-mode. The former produces colour images of blood flow and the latter a 2-dimensional greyscale image of the structure of the blood vessels.

What methods of revascularisation are available?

An endovascular approach using minimally invasive techniques such as iliac and femoral angioplasty and stenting by an interventional radiologist or invasive surgical approaches where this is not anatomically possible.

What are the main goals of surgical intervention?

The main surgical goals are to improve the patient's quality of life by eliminating or improving the symptoms of claudication, encouraging the healing of ulcers, prevention of distal amputation and ultimately, prolonged survival.

If anatomically possible (focal areas of stenotic atheroma), management with primary balloon angioplasty or stent insertion provides the most cost-effective and morbidity-free strategy, particularly in those patients most at risk from a general or regional anaesthetic.

The first BASIL-1 trial was a randomised trial comparing amputation-free survival and overall survival in patients with severe ischaemia receiving open revascularisation-first or balloon angioplasty-first revascularisation approach.

It was found that in the initial two years, there was no difference between the two groups in terms of overall survival, amputation-free survival and quality of life. However, beyond two years, there was a significant advantage of having open surgery when comparing these endpoints.

What type of vascular grafts can be used?

These can be synthetic or using the patient's own vein, typically the long saphenous. The latter is favoured for infra-inguinal revascularisation because long-term graft patency is superior to synthetic grafts.

The synthetic grafts are typically made of woven dacron (polyethylene terephthalate). These can be collagen or heparin bonded or expanded polytetrafluorethylene (ePTFE) or heparin bonded ePTFE. To reduce the chance of intraoperative and immediate post-operative occlusion by clot, prior to revascularisation, an intravenous dose of heparin is administered. A typical adult dose is 5,000 I.U.

What common procedures associated with peripheral vascular disease may present to the anaesthetist?

Much depends upon the point of the peripheral arterial system which is being bypassed and which artery is proximal and which artery is distal.

For example, a femoro-distal revascularisation might be a femoral to popliteal artery or femoral to posterior tibial artery.

Other examples include a femoral artery to femoral artery crossover graft or iliac artery to femoral artery.

A 73-year-old man presents on a vascular surgical list for an elective femoro-distal bypass graft for intermittent claudication. He has smoked 20 cigarettes for 50 years and has chronic pulmonary obstructive disease, ischaemic heart disease and hypertension. He currently takes ramipril 10 mg once daily, aspirin 75 mg once daily and simvastatin 40 mg once daily orally. He also takes salbutamol, 2-puffs via an inhaler on a prn basis.

How would you manage this patient's anaesthetic?

This clinical scenario may feature as a short-case or long-case in the SOEs. The line of questioning from the examiner will focus on the preoperative assessment and the proposed anaesthetic based on this. This would include a 'Plan A' and possibly a 'Plan B'. Questions will be directed on anaesthetic technique, drugs used, and common issues that are associated with this type of surgery. A safe postoperative management strategy is as important as the anaesthetic assessment and anaesthetic.

The aim of my preoperative assessment would be to establish the severity of the patient's comorbidities by history, examination and reviewing appropriate investigations. This would allow me to decide on the most appropriate management strategy. If necessary, I would seek advice from a Respiratory Physician or Cardiologist regarding preoperative optimisation but this should not delay the timing of surgery if the limb has critical ischaemia.

What are the general principles of anaesthesia when presented with a patient having surgery for peripheral arterial vascular disease?

The general principles would be to choose a technique that is tailored to the patient's actual and potential comorbidities. One should maintain cardiovascular stability, and avoid hypoxia, hypovolaemia and hypothermia. The use of invasive haemodynamic monitoring and postoperative care in a high dependency unit should be considered during the preoperative assessment.

Because of the peripheral location of this type of surgery, regional anaesthesia, with or without sedation is an ideal option.

What are the advantages of regional anaesthesia in these patients?

Firstly, a general anaesthetic is likely to adversely affect a patient with ischaemic, cerebrovascular and respiratory disease.

The avoidance of postoperative cognitive dysfunction (POCD) even with sedation is key to the management of the patient in the postoperative period. Regional anaesthesia can obtund the metabolic response to trauma, a particular concern if the patient is diabetic. Other advantages include, reduced blood loss, improved postoperative pain relief and a reduced chance of deep vein thrombosis.

However, some of these procedures may extend beyond the predicted length of surgery. With this in mind, the insertion of catheters into the intrathecal, epidural or a nerve sheath will allow a prolongation of the anaesthetic.

What might contraindicate a regional anaesthetic technique?

It might be difficult to manage a patient who is confused, restless or is unable to lie flat for a significant length of time. Performing a central neuraxial block on a patient taking warfarin, clopidogrel and other drugs that modify coagulation pathways need to be borne in mind. Early communication with the surgical team is essential to allow the most appropriate time to stop taking the drugs.

Local and systemic sepsis will need to be excluded.

If a regional anaesthetic is contraindicated, what general anaesthetic technique would you use?

I would choose a general anaesthetic technique that would, in theory have least effect on the cardiovascular and respiratory system. Bearing in mind, the possible length of the procedure I would use a total intravenous anaesthetic technique using propofol and remifentanyl and control ventilation via an endotracheal tube. I would monitor the patient's haemodynamic status via an arterial line. Following surgery, most patients would return to the Vascular Ward for routine monitoring. A postoperative analgesic regimen of regular paracetamol/codeine or morphine PCA/paracetamol would normally be sufficient.

Is there any difference in postoperative outcome in patients who have had a general anaesthetic compared with regional anaesthesia for infra-inguinal bypass surgery?

Currently, evidence to suggest the outcomes following regional anaesthesia compared with general anaesthesia is conflicting. However, a recent nationwide retrospective cohort study in Denmark suggested that regional anaesthesia is associated with better short and long-term outcomes.

A 75-year-old man is scheduled for a right below knee amputation for dry gangrene. He has significant rest pain requiring regular paracetamol and patient-controlled analgesia with morphine.

Amputation of toes, forefoot and above/below knee amputation stem from end-stage peripheral vascular disease but feature on vascular surgical lists.

What is the optimal method of managing this patient to prevent chronic postoperative amputation pain (CPAP)?

It is important to identify patients who are at risk of CPAP. This patient is already at risk because he is already experiencing significant preoperative pain. Other risk factors include previous surgical amputations, poor socioeconomic status, anxiety and psychiatric illnesses. He would benefit from preoperative optimisation of his pain prior to surgery to pre-emptively reduce noxious sensitisation of the central nervous system.

In addition to psychological support systems (allay anxiety, improve coping strategies, education regarding pain expectations, etc.) an aggressive multimodal analgesic regimen should be instituted.

The pre-emptive analgesic regime might include regular dosing with paracetamol, NSAIDs, gabapentin and an appropriate regional anaesthetic block while continuing his PCA.

Which peripheral regional blocks would be the most appropriate in this patient?

The ideal scenario would be to insert two catheters with ultrasound guidance into the nerve sheath of the popliteal sciatic nerve and the saphenous nerve. The placement of catheters allow the instillation of local anaesthetic by infusion in the preoperative-operative and well into the postoperative period (5–7 days). This will require regular review by the Acute Pain Team.

This multimodal analgesic approach aims to help wean the patient from opioids.

Other drugs used intraoperatively to reduce the incidence of CPAP and opioid withdrawal are intravenous sub-anaesthetic ketamine infusions and dexmedetomidine or clonidine.

A patient with long-standing diabetes presents for a right mid-foot amputation for dry gangrene on your Vascular Surgery list. Which peripheral nerve blocks would be most appropriate in this patient?

Because of the peripheral nature of the surgery my first-choice anaesthetic technique would be to perform an ultrasound-guided ankle block, targeting the terminal branches of the sciatic nerve (posterior tibial, superficial peroneal, deep peroneal and sural nerves) and terminal branch of the femoral nerve (saphenous nerve), which provide sensory innervation to the remainder of the foot.

Further Reading

Bisgaard J, Torp-Pedersen C, Rasmussen BS, Houliand KC, Riddersholm SJ. Regional Versus General Anaesthesia in Peripheral Vascular Surgery: A Propensity Score Matched Nationwide Cohort Study of 17 359 Procedures in Denmark. *European Journal of Vascular and Endovascular Surgery*. 2021; 61: 430–438.

Bradbury et al. Bypass versus Angioplasty in Severe Ischaemia of the Leg (BASIL) trial: An intention-to-treat analysis of amputation-free and overall survival in

patients randomised to a bypass surgery-first or a balloon angioplasty-first revascularisation strategy. *Journal of Vascular Surgery*. Supplement May 2010; 51; (5): 55–175.

Fraser K, Raju I. Anaesthesia for lower limb revascularisation surgery. *BJA Education*. 2015; 15, (5) 225–230.

Norgren L, Hiatt WR, Dormandy JA et al. Inter-society consensus for the management of peripheral arterial disease (TASC II). *Journal of Vascular Surgery*. 2007; 45: 55–67.

2.11.4 Interventional Radiology – James Harrison

This question could be presented as either a short or a long case. Electromagnetic radiation and radiation safety could be presented as a linked clinical sciences question.

You are asked to provide anaesthetic support to a patient undergoing endovascular coiling of an intracranial aneurysm following a subarachnoid haemorrhage. What are the relevant anaesthetic considerations?

In brief: this is neuroanaesthesia provided in a remote location with the additional risk of radiation exposure.

With the exception of hybrid theatres, almost all interventional radiological (IR) procedures requiring anaesthetic support constitute remote site anaesthesia. This is discussed elsewhere and will not be covered in detail here.

While most IR procedures are not particularly painful, they are often long, technically challenging procedures requiring long periods of absolute motionlessness and sometimes breath-holds. This may be further complicated by the effects of the underlying pathology, for example an intracranial bleed or space-occupying lesion resulting in obtundation or agitation.

With some procedures there is a risk of needing conversion to emergency open surgery, for example a ruptured intracranial aneurysm requiring decompressive craniectomy.

Large volumes of IV contrast are often required as part of IR procedures. This presents a potential risk of both nephrotoxicity and fluid overload.

Many IR procedures unavoidably use large doses of radiation. For the patient this is a large dose but a single exposure. For staff in IR, the risk is of cumulative exposure to smaller doses of radiation.

Describe your perioperative anaesthetic care for this patient.

Preoperative

Standard preoperative assessment to include anaesthetic, medical and drug history, as well as assessment of airway and starvation status.

In addition to this, history should be obtained of the current illness, any multisystem effects e.g. SAH causing arrhythmias or cardiomyopathy, and an overview of relevant imaging. Specifically for intracranial pathology, a baseline assessment should be made of GCS, pupil size and reactivity, and any focal neurological deficit.

Blood tests to include full blood count, urea and electrolytes, clotting profile and a group and screen.

Induction

Standard AAGBI monitoring with the addition of arterial monitoring; this allows for both tight blood pressure control and repeated sampling for anticoagulation if required.

Large bore IV access should be secured due to the potential risk for catastrophic haemorrhage. Central venous access should be considered if it is likely that central vasopressors will be required.

The anaesthetic machine is typically distant from the head of the patient; a reinforced or south-facing RAE endotracheal tube is easier to keep out of the way of the imaging gantry and a sufficiently long circuit should be fitted to the ventilator.

Neuroprotective strategies should be employed including the avoidance of hypoxia, maintenance of normocapnia (4.5–5.0 kPa) and normoglycaemia. Systolic blood

pressure should be maintained at 100–160 mmHg to maintain cerebral perfusion pressure while minimising the risk of rebleed. Both vasopressors and vasodilators/remifentanyl should be available.

A temperature probe should be inserted and the patient is likely to need active warming to maintain normothermia.

A urinary catheter should be inserted; procedures are typically long and involve the use of significant volumes of IV contrast.

Maintenance

The use of either inhalational or total intravenous anaesthesia is acceptable.

IR procedures are not typically stimulating but do require long periods of motionlessness. This can be achieved with either muscle relaxation or remifentanyl infusion.

Heparin is often required. A baseline ACT should be taken and 70–100 units/kg administered. The target ACT is 2–3x baseline.

Emergence and recovery

Smooth extubation with avoidance of coughing is desirable. This avoids spikes in BP and ICP with the associated risk of rebleeding and rupture of unprotected or partially protected aneurysms.

The patient will require an appropriate level of postoperative monitoring. This is typically HDU/ITU and will require intrahospital transfer of the patient.

What risk does radiation present to staff in IR?

Staff may be exposed to radiation directly from the X-ray tube, leakage through the collimator shielding and, most commonly, from radiation scatter from the patient. The X-rays used in IR produce their adverse effects on tissues via an indirect pathway; the radiation interacts with atoms and molecules to produce high-energy electrons which then collide with other molecules to form free radicals. It is these free radicals that disrupt DNA and alter the fate of the cells affected.

At the relatively low doses to which staff are typically exposed, the risk of acute radiation effects caused by cell death such as bone marrow suppression and GI upset is low. Low-dose exposure tends to cause cell damage that either results in repair or carcinogenesis.

How may this risk be minimised?

All staff should wear lead aprons and thyroid shields. The distance from the radiation source should be maximised; the radiation dose decreases with the distance from the source according to the inverse square law, which determines that the intensity of radiation is inversely proportional to the square of the distance from the source.

What regulations govern the use of radiation in healthcare?

The two key regulations surrounding the use of radiation are Ionising Radiation Regulation 1999 (IRR99) and the Ionising Radiation (Medical Exposure) Regulations 2000 (IRMER). The former refers to the conduct of any radiation employer and its employees, whereas IRMER applies specifically to medical radiation exposure. Both are enforceable under the Health and Safety Act 1974.

The main aim of both regulations is to ensure that the dose of any radiation received is as low as reasonably possible.

A patient with an acute ischaemic stroke is planned for mechanical thrombectomy in the IR suite. What is mechanical thrombectomy and what are the specific anaesthetic considerations for this procedure?

Mechanical thrombectomy involves passing a guidewire and microcatheter from a (typically femoral) access point to the location of the thrombus in the cerebral circulation. The artery may be controlled with an occluding balloon before the clot is retrieved with suction aspiration or other mechanical retrieval device.

The key considerations are ensuring the patient is still, controlling arterial pressure and allowing for timely assessment of neurological function. These are in addition to those for IR procedures in general, as outlined above.

Patients who are cooperative, not obtunded and without significant bulbar dysfunction may be managed with local anaesthetic and conscious sedation. A general anaesthetic will otherwise be required, typically using a rapid sequence induction and TIVA.

Intra-arterial pressure monitoring is standard practice. In an ischaemic stroke, the cerebral autoregulatory response results in hypertension. This can be beneficial in driving perfusion of the ischaemic penumbra, but uncontrolled systolic hypertension of $>220/110$ mmHg may be associated with an increased risk of haemorrhagic transformation and should be controlled. Patients who have been administered intravenous thrombolysis should have the arterial pressure kept $<180/100$ mmHg. CPP should be maintained at 70 mmHg which in practice means a target MAP of 90 mmHg, assuming an ICP of 20 mmHg.

It is common practice to use TIVA for patients requiring GA for thrombectomy. In combination with processed EEG depth of anaesthesia monitoring (e.g. bispectral index) this should allow for a more rapid recovery and assessment of neurological function. If inhalational anaesthesia is used, the MAC should be kept ≤ 1.0 to reduce uncoupling of cerebral blood flow and cerebral metabolic rate.

Are you aware of any clinical trials indicating what mode of anaesthesia provides the best outcomes in mechanical thrombectomy?

The SIESTA (Sedation versus Intubation for Endovascular Stroke Treatment) trial 2016 was an RCT examining the use of conscious sedation versus general anaesthesia in thrombectomy for acute ischaemic stroke. There was no difference in the primary outcome measure of NIHSS score at 24 hours.

Secondary outcomes indicated that patient movement was less common in the GA group, but that this group did have a higher rate of postoperative hypothermia, delayed extubation and pneumonia. At three months there was no mortality difference between the groups, although more patients in the GA group were functionally independent. The study authors concluded that there was not sufficient evidence to recommend conscious sedation over general anaesthesia.

Further Reading

- Patel S, Reddy U. Anaesthesia for interventional neuroradiology, *BJA Education*. 2016; 16 (5): 147–152.
- Redgrave J, et al. Interventional therapies in stroke management: Anaesthetic and critical care implications, *BJA Education*. 2017; 17(2): 43–47.
- Schönenberger S, Uhlmann L, Hacke W, Schieber S, Mundiyanapurath S, Purrucker JC, Nagel S, Klose C, Pfaff J, Bendszus M, Ringleb PA, Kieser M, Möhlenbruch MA, Bösel J. Effect of conscious sedation vs general anesthesia on early neurological improvement among patients with ischemic stroke undergoing endovascular thrombectomy: A randomized clinical trial. *Journal of the American Medical Association*. 2016; 316(19): 1986–1996.
- Taylor J, et al. Radiation safety for anaesthetists. *Continuing Education in Anaesthesia Critical Care and Pain*. 2013; 13 (2): 59–62.

Emergency Medicine

3.1.1 Acute Poisoning – Ami Jones

You are called to the emergency department (ED) to review a 29-year-old woman with impaired consciousness who is suspected to have taken an overdose of tricyclic antidepressants, benzodiazepines and paracetamol.

Describe your initial management of this patient.

Having first established some brief details of her past and present medical history, I would first assess this patient in a systematic manner concentrating on the adequacy of her airway, breathing and circulation before making an assessment of her level of consciousness using the Glasgow Coma Score (GCS).

The patient is maintaining her airway, has a respiratory rate of 20 breaths/min and an oxygen saturation of 99% on 15 l/min of oxygen via a non-rebreathing mask. Her pulse rate is 46 beats/min and her blood pressure is 90/60 mmHg.

She is opening her eyes to painful stimulation, localises to pain and is confused and disorientated. What is her GCS score?

Her GCS score is 11 (2 for best eye response, 5 for best motor response and 4 for best verbal response).

What would your next steps be in this patient's management?

Now that I have ensured her basic vital functions are maintained and she is conscious enough to protect her own airway I would try to ascertain a more precise history of which drugs were taken, and if possible, the exact doses and the timing of ingestion. I would also ask if she had been drinking alcohol.

Her mother has brought in several empty bottles of medication found at the scene. It is estimated that she has taken approximately 12 g paracetamol, 100 mg of temazepam, 1.5 g of amitriptyline and half a litre of vodka about 5 hours prior to her arrival in the ED.

What investigations would you like to order now?

I would order a full blood count, U&Es, glucose, liver function tests, coagulation screen, paracetamol and salicylate levels, an ECG and a chest X-ray. An arterial blood gas would be useful to assess her acid-base status.

Why have you have ordered a 12-lead ECG and a chest X-ray?

The patient has taken a significant amount of a tricyclic antidepressant (TCA). More than 1 g in a 70 kg adult can cause severe toxicity that often manifests with ECG abnormalities including an increased QRS duration, a long QT interval, an increased PR interval and AV block as well as ventricular arrhythmias. The chest X-ray may help to determine whether pulmonary aspiration has occurred.

Are there any other systems that can be affected by TCAs?

Many of the initial symptoms and signs are associated with anticholinergic effects including a dry mouth, mydriasis and blurred vision. The central nervous system can also be involved with agitation, lethargy, myoclonus, hyperreflexia, seizures and eventually, coma. Central nervous system depression may cause hypoventilation. The patient may present with a sinus tachycardia, arrhythmias, hypertension or hypotension.

Is there any specific treatment for TCA overdose?

The management of tricyclic anti-depressant overdose depends upon the severity of the symptoms. Treatment is mainly supportive with the administration of oxygen, fluid maintenance and treatment of hypotension, arrhythmias and seizures. Magnesium sulphate may be useful in the treatment of arrhythmias.

The patient should be managed in a high dependency area with continuous ECG monitoring.

TCAs become less protein bound if the patient has a metabolic acidosis. This increases the free fraction of the TCAs in the blood potentially enhancing their toxicity. An intravenous infusion of sodium bicarbonate will increase the pH of the blood and raise the protein bound fraction of the drug.

Would activated charcoal be of use?

Activated charcoal should only be used if less than an hour has elapsed since the tablets were ingested.

This patient has also taken 12 g of paracetamol. Is this of concern to you?

Yes. Metabolism is primarily in the liver. There are three main pathways: Conjugation with glucuronide and sulphate (70–80%) and hydroxylation. Cytochrome P450 enzyme system produces an alkylating agent NAPQI (*N*-acetyl-*p*-benzo quinone amine) that is normally irreversibly conjugated with the sulphhydryl groups of hepatic glutathione. NAPQI is hepatotoxic when hepatic glutathione is depleted; this can occur when a large dose of paracetamol is ingested.

A toxic dose after a single ingestion is 150 mg/kg and so 12 g of paracetamol is a significant dose. This patient is at significant risk of developing severe liver damage if it is not effectively treated.

What are the symptoms and signs of an untreated paracetamol overdose?

In early stages (<24 hours), the patient may be asymptomatic. Nonspecific symptoms of anorexia, nausea, vomiting and abdominal pain (right hypochondrium) may be a feature.

But if allowed to progress without specific treatment (12–48 hours): hypoglycaemia, coagulopathy, encephalopathy, metabolic acidosis and cerebral oedema may supervene.

Patients may progress to develop acute renal failure, arrhythmias and acute pancreatitis.

How should paracetamol overdose be treated?

Immediate treatment is essential in accordance with established guidelines.

If <1 hour since ingestion then activated charcoal should be given orally.

Plasma paracetamol levels should be measured at 4 hours or more after ingestion and treated with *N*-acetylcysteine (NAC) up to 24 hours after ingestion (maximal effect up to 8 hours following ingestion).

The dose of NAC is then guided by using paracetamol concentration/time since ingestion nomograms. If evidence of hepatic failure, discuss with regional liver unit.

How does NAC work?

NAC acts as a precursor for glutathione, replenishing hepatic stores and therefore enhances the conjugation of paracetamol.

What is the dose of NAC?

The 150 mg/kg IV as a loading dose over 15 minutes, then 50 mg/kg over 4 hours, then 100 mg/kg over 16 hours. This is continued for 36 hours or longer until the INR is in normal limits.

Does NAC have any side effects?

Yes, it commonly causes nausea and vomiting as well as urticaria, fever and bronchospasm.

Where should this patient be looked after?

If she had only taken paracetamol then she could be managed on a medical ward, but since the amitriptyline overdose is associated with hypotension and a reduced GCS she should be cared for in a high dependency area.

You are called to the ED to assess a 54-year-old man who has been found unconscious in a fume-filled car. He has been brought into hospital by the paramedics, who have also found a suicide note.

Describe your initial management of this patient.

I would assess the patency of the airway, the adequacy of the breathing and the status of the circulation. I would also make a rapid assessment of the patient's Glasgow Coma Score.

The patient is making grunting respiratory efforts with seesawing of his chest and abdomen, what are your next actions?

The patient is showing signs of upper airway obstruction and therefore I would apply simple manoeuvres to establish a patent airway. I may also need to insert a Guedel or

nasopharyngeal airway. I would also give the patient 15 l/min of oxygen via a non-rebreathing mask or give as close to 100% oxygen as possible.

This improves his respiratory pattern. His oxygen saturations are 100% on 15 l/min of oxygen via a non-rebreathing mask. His blood pressure is 70/40 mmHg and his pulse rate is 50 beats/min. His GCS is 3.

How would you further manage this patient?

I would establish wide bore intravenous access, commence fluid resuscitation and prepare to intubate and ventilate this patient. Given the history of entrapment in a fume-filled car, his reduced blood pressure, pulse rate and conscious level I suspect he may have severe carbon monoxide poisoning. I would send blood for routine biochemical and haematological investigations but would specifically request arterial blood gas analysis and measurement of carbon monoxide (CO) levels. I would also want to review a 12-lead ECG, chest X-ray and a toxicology screen. I would perform a rapid sequence induction, intubate his trachea and ventilate him with 100% oxygen. He may also require vasoactive drugs if his hypotension remains refractory to fluid resuscitation.

What are the symptoms and signs of acute carbon monoxide poisoning?

Carboxyhaemoglobin concentrations in heavy smokers may range from 3% to 10%. Symptoms may be experienced in the 10–30% range and death can result from higher concentrations.

Mild poisoning often presents with nonspecific symptoms of headache, nausea and visual disturbances. Moderate poisoning may present with confusion, hyperventilation, tachycardia and syncope. Higher concentrations lead to seizures, coma, hypotension, cardiac dysrhythmias, pulmonary oedema and eventually cardiorespiratory arrest.

What is the initial treatment for severe carbon monoxide poisoning?

The initial treatment is supplemental high flow oxygen at as high a concentration as is possible. Carbon monoxide's affinity for haemoglobin is 250 times greater than that of oxygen. The oxygen-carrying capacity of the blood is reduced because oxygen is displaced. The resultant anaemic hypoxia, leftward displacement of oxygen–haemoglobin dissociation curve and inhibition of mitochondrial cytochrome A3 results in cellular hypoxia and acidosis. Administering 100% oxygen reduces the half-life of CO from 4 to 6 hours to 60–90 minutes.

Are there any other treatment options?

Hyperbaric oxygen has two theoretical advantages. It reduces the half-life of CO to 23 minutes and significantly increases the oxygen content of the blood by enhancing its solubility in plasma. Only a few limited case reports have suggested that hyperbaric oxygen therapy helps to prevent permanent neurological damage.

Hyperbaric oxygen therapy should be considered in patients who have moderate to severe CO toxicity associated with neurological impairment. There are relatively few hyperbaric oxygen chambers in the United Kingdom. This has anaesthetic implications for managing a critically ill patient on a long-distance transfer and then in an unfamiliar environment.

What other toxic gas is associated with smoke inhalation?

Cyanide also causes cytotoxic hypoxia. It is released when materials such as wool, silk and plastic undergo combustion. It is common in household and industrial fires.

What are the symptoms and signs of cyanide poisoning?

Mild toxicity can cause dizziness, headache, drowsiness and dyspnoea with progression to confusion, agitation and seizures. Eventually coma and cardiorespiratory arrest may follow.

How would you manage a patient who presented with cyanide toxicity?

The treatment options are initially supportive but specific antidotes can then be considered. I would administer 100% oxygen to the patient and if necessary, intubate and control ventilation. Hypotension can be managed with fluid resuscitation and vasopressor agents. I would send a blood sample for cyanide levels, blood gases and lactate concentrations. There is likely to be a severe metabolic acidosis.

There are a number of specific antidotes available, which vary depending on which country you are working in. In some countries a cyanide antidote package exists which consists of sodium and amyl nitrates and sodium thiosulphate. The nitrites oxidise haemoglobin to methaemoglobin, which has a high affinity for cyanide and forms cyanmethaemoglobin which the liver then metabolises.

A more commonly used and less complicated antidote is hydroxycobalamin. Hydroxycobalamin displaces cyanide from cytochrome oxidase in the mitochondria and forms cyanocobalamin, which is then excreted.

Do you know of any treatments given in a hospital environment that can actually cause cyanide toxicity?

Rapid or prolonged administration of the vasodilator sodium nitroprusside (SNP) can cause toxicity significant enough to cause a metabolic acidosis. The metabolites of SNP include cyanide ions. Prolonged or high-dose sodium nitroprusside therapy can therefore lead to iatrogenic cyanide poisoning.

Further Reading

Joint Formulary Committee. *British National Formulary*. 58th edition. London: British Medical Association and Royal

Pharmaceutical Society of Great Britain, 2009.

Ward C, Sair M. Oral poisoning: An update. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2010; 10(1): 6–11.

3.1.2 Burns, Electrocution and Drowning – Helen L Jewitt, Philip Harrington and Stephanie Wallis

These are potentially life-threatening situations, the initial management of which you will be expected to be confident of. Apply a structured approach and add the details specific to the particular scenario you are given.

A 25-year-old male is brought into A and E following a house fire.

Describe your initial assessment.

I would use a structured approach to make a rapid initial assessment of the patient. This will identify potentially life-threatening problems and make a preliminary estimation of the extent of the burn. Cervical spine control must be maintained if there is any history of trauma. I would simultaneously obtain a brief history either directly from the patient where possible or a collateral history from emergency personnel present at the scene. This history would include the timing and circumstances of the injury, consumption of alcohol or recreational drugs and relevant past medical history.

I would firstly assess the patency of the airway looking for signs of airway burns including visible burns to the face, oropharyngeal oedema and singed hair or soot in the nostrils. I would listen for hoarseness or stridor which indicate airway compromise. High flow humidified oxygen should be given via a non-rebreathing bag and facemask.

I would make an assessment of the respiratory system by observation of the chest for respiratory movements and involvement in the burn, determination of respiratory rate, auscultation of the chest and measurement of oxygen saturations.

Circulation would be assessed by feeling the warmth of the peripheries and measuring central capillary refill time, heart rate and blood pressure. I would obtain intravenous access with two large bore cannulae, ideally through unburnt skin, and commence fluid resuscitation.

Assessment of the neurological system would consist of determination of Glasgow Coma Score or AVPU score and assessment of the pupils. I would titrate intravenous morphine to the patient's pain provided there were no contraindications.

The patient should then be exposed to allow the extent of the burn to be estimated while maintaining core body temperature.

How is the severity of a burn graded?

Burns can be graded both on the depth of the injury and the percentage surface area that is affected. The depth of a burn can be subdivided into superficial or deep. Superficial burns affect the epidermis only. They appear red, can blister and are painful. Deep burns cause damage to the dermis and have a white appearance. There is loss of pinprick sensation due to destruction of the nerve endings.

Percentage surface area can be estimated using the 'rule of nines', whereby areas of the body are assigned a surface area based on multiples of nine. This rule is not applicable to children as they have small limbs in comparison to their head and trunk. Alternatively the area of the patient's own palm can be used to represent 1% of their total surface area.

What further investigations are appropriate for this patient?

Blood tests should be performed including full blood count and urea and electrolytes. Alcohol levels and drug toxicology may be indicated depending on the history. Arterial blood gases should be obtained. Co-oximetry allows accurate determination of carboxyhaemoglobin levels. A chest X-ray should be performed at this stage.

What would alert you to the possibility that this patient may have suffered an inhalation injury?

Features from the history and clinical examination would raise the possibility of an inhalational injury. In the history, evidence of prolonged exposure to smoke such as entrapment in a burning building or loss of consciousness at the scene are worrying features.

Evidence on examination of soot round the nostrils or inside the mouth, facial burns, hoarseness, respiratory distress, stridor, soot staining of the sputum or oropharyngeal oedema are markers of possible inhalational injury.

What is the appropriate course of action if inhalational injury is suspected?

The airway should be secured by endotracheal intubation as a priority. I would anticipate a potentially difficult airway and ensure I have appropriately prepared for this. Swelling of the airway can progress rapidly leading to complete obstruction. A range of endotracheal tube sizes should be immediately to hand as the oedema may be advanced by the time of presentation to hospital. An un-cut endotracheal tube should be used to allow for the development of facial oedema and one may consider using a reinforced tube to prevent occlusion or kinking. A nasogastric tube may be inserted at the same time to permit early enteral feeding.

How is the fluid requirement of this patient calculated?

The most commonly used equation for estimating the fluid requirement following a major burn is the Parkland formula. The information needed to make the calculation is the patient's weight, the percentage area of the burn and the approximate time of the injury.

Over the 24 hours after the injury the patient should receive 4 ml/kg of crystalloid solution per percentage area of the burn. Half of this total volume should be administered in the first 8 hours after the burn and the remainder in the following 16 hours. This formula only provides guidance for fluid administration and additional fluid may be required.

What specific monitoring and investigations are indicated in the first 24 hours of admission following a major burn?

The patient should be managed in a critical care environment.

The airway is maintained by endotracheal intubation with adequate sedation.

Invasive monitoring is indicated to assess the adequacy of ventilation and the response to fluid resuscitation. A urine output of 0.5–1 ml/kg/hr should be ensured. Haematocrit, urinary osmolality and serial plasma electrolytes can be used to guide fluid replacement.

A full secondary survey is performed to detail the full extent of the burn and any other injuries. Circumferential burns which compromise ventilation or distal perfusion require escharotomy.

Strict attention should be paid to analgesia, thromboprophylaxis, gastric ulcer prophylaxis and nutrition.

What are the criteria for transfer of this patient to a regional burns unit?

Discussion should be initiated with a regional unit and transfer considered if this patient has a burn surface of more than 10% of total body surface area, has sustained burns to specific parts of the body including face, hands, feet, genitalia, perineum or major joints; or there is an inhalational injury.

Other criteria for transfer are greater than 5% surface area burns in children, electrical and chemical burns, circumferential burns to the limbs or chest and burns in patients with pre-existing medical conditions or at the extremes of age.

The patient has been transferred to a regional burns unit. He has a total burn surface area of approximately 20% affecting his right arm, flank and thigh. He does not have a significant inhalational injury.

Outline your perioperative management of this patient for debridement and split skin grafting of his burn.

Answer this in a systematic way beginning with aspects of your preoperative assessment, followed by intraoperative and postoperative issues. It is vitally important to mention an awareness of the possibility of massive blood loss, maintenance of the patient's temperature and provision of adequate analgesia.

In my preoperative assessment of this patient I would look at events since his admission to the burns unit to establish how stable he has been. In addition to this I would gather available information regarding past medical and anaesthetic history, medication and allergies. Appropriate preparation for this case includes warming the theatre to 28–32° C and ensuring there is cross-matched blood available.

I would intubate the patient ensuring that suxamethonium was not used. This is because suxamethonium can precipitate a marked increase in plasma potassium levels due to a proliferation of extrajunctional acetylcholine receptors following a burn injury. It is commonly considered to be unsafe to use suxamethonium from 48 hours after the burn for a period of up to one year.

If not already in use, invasive arterial monitoring is appropriate for this case. An arterial line allows beat-to-beat monitoring of blood pressure and regular sampling of blood gases.

One of the potential problems I would anticipate in theatre is massive blood loss and cardiovascular instability. Debrided tissue bleeds significantly and it can be difficult to keep an accurate track of how much blood has been lost. Arterial blood gases at regular intervals will show a trend in haemoglobin and point-of-care coagulation tests provide useful information. Packed cells should be transfused as indicated by the clinical findings and haemoglobin. Cardiac-output monitoring is useful to optimise fluid administration.

The patient's temperature should be monitored and they should be actively warmed with a forced air device and fluid warmer.

The postoperative care of the patient will take place on a specialised burns intensive care unit or general ITU. Strict attention should be paid to fluid balance, analgesia, thromboprophylaxis, nutrition and prevention of infection.

The patient will need regular dressing changes for which they may require further general anaesthesia or sedation and analgesia.

You are asked to see a 30-year-old electrician in the emergency department who has been injured by some faulty cabling at work. He was found on the other side of the

room by the paramedic team, has regained consciousness and is complaining of severe pain in the arm that he was using to work with.

This topic may present as part of the burns section of intensive care or may be a smaller discussion when talking about a major trauma case. It is difficult to talk about electrical burns without mentioning the mechanism by which they occur and then subsequent multisystem injuries as they are very pertinent here.

What is an electrical burn?

Electrical injury is a rare but significant form of injury sustained when the body comes into contact with a source of electricity. An electrical burn is the cutaneous injury caused by the exposure of the body to electrical current that is the result of electrical energy being transformed to thermal energy. Injuries can range from simple local erythema through to full thickness skin injury but importantly, due to the possibility of electrical current being retained by bones with high resistance, deep tissue injury can occur sparing the skin. Therefore skin injury cannot be used as a reliable assessment of internal injury in the way that it can with flame burns.

How may electricity cause injury?

Importantly the severity of the electrical injury is determined by the current intensity utilising Ohm's law. Body areas with low resistance will experience a higher current intensity for the same voltage than those with high resistance.

Electricity causes injury by direct and indirect mechanisms. The direct effects involve the effects that the electrical current has on parts of the body, for example ventricular fibrillation of the myocardium, or as mentioned above the transformation of electrical to thermal energy causing tissue burns. Indirect effects are usually the result of sustained muscle contraction brought about by the electrical current and can include rhabdomyolysis and renal injury.

Electrical injury is a multisystem problem with several organ systems affected. Cardiovascular manifestations include direct myocardial damage via muscle necrosis and also arrhythmias. Cutaneous manifestations through burns are discussed above but muscle necrosis and rhabdomyolysis may occur if extensive enough. Neurologically, current delivery to the brain can result in damage to the respiratory control centre causing respiratory arrest which can lead to cardiac arrest and its associated issues if not managed promptly. Seizures may also occur. The peripheral nervous system may also be affected via burns, scarring, vascular injury or generalised swelling.

How may the management differ from other types of burn injury?

The electrically injured patient should be treated along the same pathway as any major trauma patient with simultaneous assessment and treatment alongside a dedicated trauma team. Invasive monitoring and in particular close cardiac monitoring will be required. It should be remembered that these patients may have other major traumatic injuries on top of their electrical injury. Other specific injury patterns to assess and treat include arrhythmias from current delivery, hypoxic cardiac arrest due to tetany or brain involvement, rhabdomyolysis and its subsequent effects and subcutaneous injury. In the electrically burnt patient the degree of subcutaneous necrosis may be hidden by intact skin and it is important to involve a burns specialist when calculating fluid requirements as they may have higher requirements than a patient with thermal burns. Similarly, the possibility of a hidden compartment syndrome should be evaluated.

Are there any other issues to consider in electrocution injury?

Electrical injury may cause multiple casualties, such as in the context of a lightning strike, and these patients should be triaged appropriately. Other nuanced considerations include examining for other hidden injuries such as spinal cord injury, instituting nutritional support as in any other patient suffering major burns and the need for detailed ophthalmic and otoscopic examination as these areas may be affected by high voltage injury.

An 18-year-old man is brought into A and E having fallen into a river on an outdoor pursuits course. He is unconscious with no obvious external injuries and has a weakly palpable carotid pulse. His rectal temperature is 28 degrees.

This is an uncommon scenario but can be simplified by using advanced life support guidelines as the mainstay of your answer. Always remember and make clear in your answer that other injuries can coexist in this type of patient. Appropriate precautions must be taken, for example cervical spine protection.

What is the initial management of this patient?

After a rapid assessment of airway, breathing and circulation, the first step is to intubate the patient with cervical spine control. He should be ventilated with 100% oxygen. His cardiovascular system should be assessed for evidence of a spontaneous circulation. If this is absent, cardiopulmonary resuscitation according to the advanced life support guidelines must be commenced. Resuscitation guidelines recommend adrenaline is withheld until the patient's temperature is over 30°C. Intravenous access should be secured and administration of intravenous fluids commenced.

This patient has severe hypothermia and active rewarming measures should be commenced. These include warmed inspired gases, forced air warming blankets, warming of intravenous fluids and irrigation of the bladder or stomach with warm fluid. More invasive methods include peritoneal lavage with warm fluid and the use of extracorporeal warming.

Can you list any predictors of patient outcome following a near drowning?

There are a number of factors which are poor prognostic indicators in this type of presentation. These include a prolonged immersion time of greater than 5–10 minutes, cardiac arrest at the scene, dilated unreactive pupils or pH less than 7 on arrival to hospital and lack of purposeful motor response at 24 hours. Hypothermia, however, can be neuroprotective in the event of a prolonged resuscitation and therefore a period of observation for prognostication is often undertaken.

Further Reading

Bishop S, Maguire S. Anaesthesia and intensive care for major burns. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2012; 12(3): 118–122.

Carter E, Sinclair R. Drowning. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11(6): 210–213.

Deakin C et al. Special Circumstances Guidelines. Resuscitation Council UK. 2021.

Koumbourlis AC. Electrical injuries. *Critical Care Medicine*. 2002; 30(11): 424–430.

McCann C, Watson A, Barnes D. Major burns: Part 1 – Epidemiology, pathophysiology

and initial management. *BJA Education*. 2022; (3): 94–103.

McGovern C, Paxty K, Paton L. Major burns: Part 2 – Anaesthesia, intensive care and pain management. *BJA Education*. 2022; (4): 138–145.

Royal College of Emergency Medicine. Learning: Electrical injuries. Available from: www.rcemlearning.co.uk/reference/

[electrical-injuries/#1569236193419-972739b7-32a4](https://www.sccm.org/getattachment/50ab9630-4902-4d32-ad4e-47e531942b4e/Critical-Care-of-the-Burn-Patient).

Sheridan R. Critical care of the burn patient. *Society of Critical Care Medicine*. 2015; available from www.sccm.org/getattachment/50ab9630-4902-4d32-ad4e-47e531942b4e/Critical-Care-of-the-Burn-Patient.

Intensive Care

3.2.1 The High-Risk Surgical Patient – Matt Thomas

This topic would be most likely to appear as part of either a long or short clinical case, as it allows ample opportunity to discuss common clinical problems.

Have you any idea of mortality rates after surgery in the UK?

Data from National Confidential Enquiry into Patient Outcome and Death (NCEPOD) puts 30-day mortality somewhere between 0.7% and 1.7% for all operative procedures. About 1 in 8 patients were in a high-risk group that had a crude mortality rate after all surgery of 12.5%. Interestingly, in absolute terms, this group accounted for over 80% of the total number of deaths, but fewer than 15% of them were admitted to an ICU. The European Surgical Outcomes Study found a higher rate of 3.6% hospital mortality in the UK. Both studies are a decade old now and the Perioperative Quality Improvement Programme (PQIP), provides more current information on complications and quality of life but not yet on mortality after surgery in the UK. It is important to say that major morbidity is at least 10 times higher than mortality and has a significant impact on quality of life and healthcare costs extending well beyond the index surgical admission.

Which groups of patients have higher mortality rates?

Most important are patient- and surgery-related factors. NCEPOD have shown that mortality increased with age: patients with an age of more than 70 have 25% of all operations but suffer 75% of all deaths. Existing comorbidities, especially heart failure, and current physiological status are also important. With regard to surgical factors, mortality increases with emergency surgery and with major and major-plus procedures (i.e., laparotomy or thoracotomy). These operations are generally longer and result in more tissue damage. Anaesthetic-related factors might influence postoperative outcome. There are data to suggest that fluid management, and analgesic technique, for example, influence mortality in high-risk patients. Finally hospital-level factors such as staffing and critical care resource can make a difference.

How would you define high-risk?

Risk is difficult to define and means different things to different people, so this is somewhat subjective. Practically speaking, a common threshold is to consider a patient or procedure high-risk if mortality is estimated at over 5%, or twice the usual mortality associated with that procedure. More NCEPOD data indicate that surgeons miss 1 in 3 high-risk patients if asked to identify them preoperatively. Again, this does not take into

account the risk of major complications such as MI, kidney injury or postoperative cognitive dysfunction.

So how can these patients be identified beforehand?

It would be better to use more objective means of assessment than personal opinion. There are a number of assessment and prediction tools that can be applied in the preoperative clinic or at the bedside to estimate risk. Both patient factors and surgical risk need to be evaluated, but the latter should be easier as there are more national data on complication rates including mortality available to use as a benchmark.

What assessment and prediction tools do you know?

There are checklists and scoring systems based on a clinical history, examination and simple laboratory tests, and then there are those that involve more sophisticated investigations. Cardiac focussed investigation and risk prediction like the Goldman or Lee index has given way to more holistic estimates of all cause mortality and morbidity such as POSSUM that incorporate surgical factors. In some cases, like the National Emergency Laparotomy Audit (NELA) model or Euroscore, this might be surgical speciality specific whereas other tools like Surgical Outcome Risk Tool (SORT) or National Surgical Quality Improvement Programme (NSQIP) cover a broad range of procedures. There is also more understanding that overall physiological reserve is critical which has led to increasing use of dynamic tests of the cardiorespiratory system like 6 minute walks, or cardiopulmonary exercise (CPET) testing. This is perhaps more objective than an assessment of clinical frailty although the idea behind is similar.

Please explain your last comment a bit more.

What is seen as more important is the functional ability of the heart especially when it is put under stress, not merely the presence or the absence of coronary artery disease. Even the Goldman index reflects this, as the highest weighting for any score is for heart failure. But the heart does not work alone and lung function and muscle strength and bulk are also important in determining reserve. So a subjective assessment of frailty, using the Clinical Frailty Scale for example, is an estimate of overall physical capacity, but assessing walking or cycling ability allows this to be quantified. Individual patient risk depends on individual physiological reserve to deal with an increased demand on the body and this cannot be assessed by history and clinical examination alone.

Why identify these patients preoperatively?

Firstly, the patient can be given accurate information about the likely risks of the procedure and secondly, additional resources can be used to reduce these risks and increase the chances of survival. These resources, for example ICU beds, are expensive and scarce so must be used to greatest effect.

What do you understand by the terms 'optimisation' and 'goal-directed therapy'?

Goal directed therapy is a term used widely in anaesthesia and critical care that usually refers to more-or-less protocolised care aimed at achieving a set of predominantly

haemodynamic goals that have been associated with improved outcome in a clearly defined group of patients. Optimisation in the context of perioperative care refers to interventions that are tailored to the patient to reduce perioperative risk to the minimum possible under the circumstances.

What is the principle behind goal directed therapy?

It was first noticed over 50 years ago that patients who could not increase their cardiac output postoperatively had a higher mortality, and in the 1980s, was noted again by Shoemaker in trauma patients. It was thought that cardiac ischaemia and organ failure was caused by low cardiac output and reduced oxygen delivery, so it was hypothesised that increasing cardiac output and oxygen delivery to the levels attained by survivors would lead to reduced mortality. While Shoemaker was able to show that achieving oxygen delivery goals with oxygen, fluids and inotropes significantly reduced postoperative mortality his findings have not been consistently replicated.

So what is current thinking?

The modern concept of goal directed therapy also concentrates on oxygen supply-demand imbalance, but less on cardiac ischaemia. The stress response to surgery imposes the extra oxygen demand for adequate mitochondrial generation of ATP, required primarily for wound healing and the postoperative inflammatory response. This is met by increasing cardiac output and oxygen extraction. Patients with the inability to respond to this increase in demand are those who are at risk of developing complications because mitochondrial function is affected by prolonged reductions in oxygen delivery. The patients are therefore at risk of developing not only postoperative myocardial ischaemia but also injury to other organs like the brain and kidneys. Mitochondrial dysfunction may lead to immunosuppression and is thought to underlie multiorgan failure. It also explains why heart failure is so significant, why lower central venous saturation identifies a high-risk group, and why functional tests like CPX testing are good at predicting postoperative complications. That said the evidence for goal directed therapy in the high-risk surgical patient is still debated and trials such as FLuid Optimisation – in Emergency LAParotomy (FLO-ELA) are ongoing.

For further details on CPX testing see Section 1.2.1. 'Preoperative assessment and management of patients with cardiac disease'.

And tell me more about optimisation.

Optimisation refers to preoperative preparation that is intended to minimise risk for the patient. This can include specialist review and treatment of comorbidities, stopping smoking and drinking alcohol, exercise (so called prehabilitation), treatment of anaemia and addressing poor nutrition. In urgent cases time is much more limited but even then fluid status, anaemia, glucose control and other physiological parameters can be addressed. Only in true emergencies such as a ruptured abdominal aortic aneurysm is optimisation not really possible. However, as with goal directed therapy there is not clear evidence that optimisation clinics for elderly patients outweigh the costs.

You are asked to anaesthetise an 81-year-old woman who has presented for an elective abdomino-perineal resection. She has chronic obstructive pulmonary disease

and is known to have moderate left ventricular dysfunction. The results of a CPX test show an anaerobic threshold (AT) of 10.5 ml/kg/min without ischaemia.

What are the anaesthetic implications of this result?

It is worth being familiar with NICE guidance so that the relevant recommendations can be included in your answer. Remember though when answering case-based questions it is better to describe what you would actually do rather than refer to techniques you are not familiar with.

The combination of a major operative procedure and an AT of <11 ml/kg/min indicate that the patient has a high risk of morbidity or mortality. In the first instance an individual risk assessment should be produced using a tool like SORT to give a better picture of likely and possible outcomes to frame the discussion with the patient. The option of non-surgical management should be considered and effort made to optimise medical therapy, anaemia, and nutrition in a reasonable time frame to allow expeditious surgery. An enhanced recovery pathway should be offered even with a planned post-operative destination of a high dependency unit.

Perioperatively I would plan to use cardiac output monitoring, as although there appears to be no clinically important difference in mortality there does seem to be a reduction in complications overall. I would use this alongside arterial and urinary catheters for haemodynamic monitoring.

What cardiac output monitoring would you use?

There is no evidence to suggest that one cardiac output monitor is any better than another, and alternatives such as oesophageal doppler or pulse contour analysis using lithium dilution (LiDCO™) are available. I prefer the latter. But cardiac output is only a part of the story and I would consider heart rate, peripheral perfusion, lactate, urine output and blood pressure in the overall assessment of cardiovascular status.

So, what sort of targets are you aiming for?

Roughly normal! If the blood pressure and heart rate are around baseline, capillary refill is normal and the patient is warm and passing urine then I would be happy. In terms of blood pressure I would aim for within 10% of the preoperative value and use the cardiac output monitor to help decide whether fluid, inotropes or vasopressors are best suited to the situation.

Are there any problems associated with goal directed therapy?

As with any medical intervention there are risks and benefits. There are risks associated with the insertion of intravascular catheters, with the fluids and drugs used, and with use of the cardiac output monitoring device itself. The main problem seen with the drugs is tachycardia, which increases myocardial oxygen demand, and this is a common reason for failure to achieve the goals. There are risks with the technique applied, particularly if a pulmonary, artery (PA) catheter is used. If the cardiac output monitors are incorrectly set up or not calibrated, the data produced may be unreliable or misinterpreted. Finally, if goal-directed therapy is started too late, that is after organ failure is established, complications may be increased.

Why do you think goal directed therapy is not more widely practised?

There are three main reasons. First, there is confusion because of conflicting trial evidence and some scepticism about positive trials relating to their methodology and therefore validity. It is also worth noting that surgical and anaesthetic techniques have changed since much of the research was done which may again reduce validity. Second, there is the problem of a limited number of critical care beds, both ICU and HDU, although it is possible to use them for a short period of time in theatre and recovery areas only. Finally, there is reluctance to use PA catheters or other cardiac output monitoring devices, which may also be affected by availability of and familiarity with the appropriate equipment in theatres, or a perception that the risks outlined outweigh any benefit from a monitoring technology.

What about other drugs? Statins or beta-blockers for example?

There are theoretical advantages to both in reducing perioperative risk, but once again there is insufficient evidence to support de novo use. Patients who are already taking these drugs should continue on them and patients who are not should not have them started for any perceived risk reduction but only for accepted indications such as a postoperative myocardial infarction.

What about ventilation during the operation?

Postoperative pulmonary complications are a major component of morbidity and for this patient with COPD there is additional risk. Intraoperative ventilation can affect the incidence of postoperative pulmonary complications. I would titrate tidal volume to 6–8 ml/kg of ideal body weight as long as the driving pressure was 15 cm of water or less and would set a fairly low level of PEEP, something like 5 cm of water, given her obstructive lung disease. There's no need to aim for perfect blood gases. Using an FiO₂ to achieve saturations of 92% and a respiratory rate to achieve a pH of 7.3 would be sufficient.

And after the operation?

One of the advantages of postoperative care in a high dependency environment is the ability to provide non-invasive ventilation if required. In this case, with extensive abdominal surgery and a history of COPD, there is an argument for prophylactic use rather than waiting for respiratory failure. But just as important is early mobilisation and chest physiotherapy, both of which rely on good analgesia.

Ah, good, I am glad we have got round to that. How would you provide analgesia?

This is just one example of an analgesic regimen. As above describe what works for you in your practice and be prepared to justify your approach.

Preoperatively I give paracetamol, intraoperatively fentanyl and morphine with a small bolus of ketamine when closing. I would also ask the surgeons to place rectus sheath catheters and load with local anaesthetic. Postoperatively I would use a multi-modal approach with regular paracetamol, rectus sheath local anaesthetic infusion and oral tramadol and morphine as required. If this was insufficient I would escalate to a

morphine PCA, assuming normal renal function. With the history of heart failure I would be cautious with NSAIDs.

What do you understand by enhanced care after surgery?

This is different to the enhanced recovery after surgery pathways that have been common for the last decade or so. Enhanced perioperative care refers to a pathway for patients who fall into the ever widening gap between ward environments and critical care. Their needs cannot be met on a normal ward, but do not require the staffing and technology available in ICU. Examples might be enhanced monitoring such as of a free flap or an epidural infusion, or treatment of difficult to manage pain. It is also suitable for patients who might be at increased risk of death but who do not meet the usual definition of high risk.

Further Reading

Faculty of Intensive Care Medicine. Enhanced perioperative care. The Faculty of Intensive Care Medicine (ficm.ac.uk) accessed 24 August 2022.

Meier A, Hylton D, Schmidt U. Intraoperative ventilation in the high-risk surgical patient. *Respiratory Care*. 2021; 66: 1337–1340.

National Confidential Enquiry into Patient Outcome and Death. Peri-operative care: Knowing the risk (2011). www.ncepod.org.uk/2011poc.html accessed 23 August 2022.

National Institute for Health and Care Excellence National Guideline 180. Perioperative care in adults. 2020. www.nice.org.uk/guidance/ng180 Accessed 23 August 2022

Parker T, Brealey D, Dyson A, Singer M. Optimising organ perfusion in the high risk surgical and critical care patient: A narrative review. *British Journal of Anaesthesia*. 2019; 123: 170–176.

Perioperative Quality Improvement Programme <https://pqip.org.uk/content/home> accessed 24th August 2022.

3.2.2 Management of Multiorgan Failure and Scoring Systems in the Critically Ill – Matt Thomas and Philip Harrington

This topic may follow on from a discussion about multiorgan failure or may appear as a starter question for a discussion on other areas of ICU.

What do you understand by the term multi-organ failure (MOF)?

The term is associated with critically ill patients in the ICU. In one sense, it just means failure of more than one organ, but is usually used in the context of acute severe illness and refers to new organ failures requiring advanced organ support techniques, for example ventilation and haemofiltration. There is no one accepted definition of multi-organ failure, and it can be difficult to decide if, say coagulopathy and thrombocytopenia should be considered as liver or blood failure for example.

Do you think this is a useful concept?

The term is very non-specific, and really says nothing detailed about the cause of the problem, the organs involved and the severity, or the overall prognosis. Organ failure itself can be difficult to define, as it is often a matter of degree, not an all-or-nothing phenomenon.

Table 3.2.2 The SOFA scoring system

	1	2	3	4
Respiratory: PaO₂/FiO₂ (mmHg)	<400	<300	<200 and mechanically ventilated	<100 and mechanically ventilated
CVS: MAP or vasopressor	MAP < 70 mmHg	dop ≤ 5 or dob (any dose)	dop > 5 OR epi ≤ 0.1 OR nor ≤ 0.1	dop > 15 OR epi > 0.1 OR nor > 0.1
CNS: GCS	13–14	10–12	6–9	<6
Coagulation: Platelets (×10³/ mm³)	<150	<100	<50	<20
Hepatic: Bilirubin (mg/dl)	1.2–1.9	2.0–5.9	6.0–11.9	>12.0
Renal: Creatinine (mg/ dl) (or UO)	1.2–1.9	2.0–3.4	3.5 – 4.9 (or <500 ml/day)	>5.0 (or <200 ml/ day)

Are there any more specific descriptions of organ dysfunction?

There are two ways of looking at individual organ dysfunction. First, there are definitions that do reduce it to a simple question of failure or no failure, for example brain failure or delirium that is either present or absent. Then there are scores which recognise that organ failure is a matter of degree, for example the difference between acute lung injury and adult respiratory distress syndrome (ARDS) using the PaO₂ : FiO₂ ratio. ARDS is likely if the ratio is <200 and acute lung injury <300.

Further examples include the Child-Pugh score for liver dysfunction or the GCS for conscious level.

There are consensus definitions of organ failure, produced by European and American societies of intensive care medicine, as part of the updated sepsis definition in 2001, but these tend to focus on either the presence or absence of failure of organs. However there are elements of organ dysfunction included in prognostic scores such as acute physiology and chronic health evaluation (APACHE).

Have you heard of any composite scores of organ dysfunction relating more directly to multi-organ failure?

Sepsis-related organ failure assessment (SOFA) is a well-established and validated scoring system that was developed in Europe in the mid-nineties (Table 3.2.2). It uses daily assessments of function in six organ systems and assigns a value to each depending on the deviation from normal, where higher numbers mean worse function. The advantage of SOFA is that the progress of organ dysfunction can be followed from day to day, both overall and within each of the six organs monitored. It has been shown that mortality is related to the total score as well as the change in the score, where a

particularly poor prognostic sign is an increase in the SOFA score after the first 24 hours. These scores are increasingly used as outcome measures in clinical trials in the ICU.

What are the drawbacks of the SOFA scoring system?

This scoring system works well, but there are a number of issues. First, there is the issue of case mix. For example, the SOFA score was developed by consensus to apply to septic patients and must not be assumed to apply to all critically ill patients, particularly in specialist areas, although in fact, validation of SOFA score has been performed in populations of trauma, cardiac surgery and bone marrow transplant patients among others, and appears robust in each. The problem of lead-time bias is less with daily scores than with the general prognostic scores that use only admission data.

It is not developed as an outcome measure tool and there is some scepticism regarding the relevance of a sudden fall in the SOFA score must be maintained. Also there does not seem to be a simple bedside way of quantifying cardiac dysfunction independent of therapy. SOFA currently uses dose of vasoactive drugs, but in future, the measurement of brain B-natriuretic peptide and troponins might aid prognostic scoring.

What causes multi-organ failure?

Multi-organ failure is seen with all the illnesses that present to intensive care – medical, traumatic and surgical. It is the endpoint of a severe systemic inflammatory response whatever the trigger. When it develops after some time in the ICU the most likely trigger is infection.

Is there a final common pathway at cellular level?

There are several theories of the origin of cell dysfunction in multi-organ dysfunction, which may just reflect the large number of triggering events rather than the cell processes responsible for organ failure which are more likely to be stereotypical (this is not clear). Examples of triggers include sepsis, trauma, or ischaemia especially with reperfusion injury. Each induces a pro-inflammatory response that includes local and systemic leucocyte and endothelial activation and activation of innate immune and coagulation systems.

So how does this actually lead to organ failure?

This process leads to microvascular occlusion within organs that in turn results in cellular hypoxia and dysfunction even if global haemodynamics and oxygen transport appear relatively normal. There is certainly evidence of microvascular dysfunction in systemic inflammatory response syndrome (SIRS) and multi-organ failure especially secondary to sepsis. One step further, others have noted the lack of evidence of infarction in affected organs and their potential for complete recovery, and have proposed that multi-organ failure is a form of adaptive cellular hibernation triggered by periods of cytopathic hypoxia (diminished ATP production despite normal oxygenation) maintained by the hormonal and metabolic changes induced by systemic inflammation. What has not been explained is why some develop multi-organ failure and others do not, and why a particular organ might be affected in one individual and not another.

What treatments are available for multi-organ failure?

Treatment of the cause, if it can be identified, is essential, but as yet there are no specific treatments for multi-organ failure, although Singer's hypothesis of hibernation may generate some interesting therapies. At the moment the focus is on prevention, support and avoiding secondary insults. Ensuring adequate early resuscitation and control of the trigger for SIRS are the basis of prevention. Maintaining oxygen delivery to mitochondria early in the critical illness in theory prevents the "switch off" that underpins cell and organ dysfunction. If MOF does occur then the patient must be supported until recovery of function occurs. In this period avoidance of secondary insults, notably infection, but also hypoxia, hypotension, hyperglycaemia, will reduce the period of organ failure.

Is this true in every case?

There is the odd situation, such as fulminant hepatic failure, where single-organ transplantation is a treatment for multi-organ failure, but the general principle of supporting organs and maintaining homeostasis does apply to all.

What specific organ support techniques are available?

Mechanical devices are available to support lungs, heart, kidneys and liver although some are more effective than others, or more suitable for long-term use. Drugs are also available for support of cardiovascular function, with there being no really effective pharmacological support of other organs.

The gut may be supported by enteral or parenteral feeding or drug therapy of motility disorders. Blood products, erythropoietin, vitamin K and haematinics are used to support haematology and coagulation, and dressings and grafting to protect the skin. Endocrine support currently consists of insulin and glucocorticoids in selected patients, although this may be an area that can be developed in the future. There is no substitute for the brain, although a range of drugs can be used to alter function, usually depressing it to reduce oxygen demand (CMRO₂) thus protecting against ischaemia.

Are these support techniques benign?

Drug therapies have side effects that may be unpredictable in the context of critical illness, or there are unintended consequences. For example, iron supplementation may help prevent anaemia and avoid transfusion, but the same iron may promote bacterial overgrowth. Catecholamines are widely used for haemodynamic support, but increase myocardial oxygen demand, have metabolic consequences and enhance bacterial growth. Blood products carry risks of immunosuppression, incompatibility and infection. Catheters are associated with complications of insertion, infection and if intra-vascular, thrombosis. Ventilation, the supportive therapy that distinguishes intensive care, has the potential to cause further lung injury when used inappropriately. It is also often forgotten that many interventions in ICU are painful if nothing else. Before starting any therapy the benefits, risks, alternatives and consequences of doing nothing must be considered.

Are there any other important adjuncts to supportive therapy?

DVT and stress ulcer prophylaxis are essential in the absence of contraindications. Good nursing care and physiotherapy are also important in general supportive care. Infection

control and good hygiene are vital, as patients with MOF have increased susceptibility to infections.

What is the mortality associated with multi-organ failure?

It is very difficult to predict individual outcome, and models such as APACHE scores are intended for population and not personal use. A very rough guide is approximately 20% risk of mortality per organ failure, although this will vary with the cause, co-morbidity, organs failed and initial response to therapy.

Are there any particularly ominous signs?

Some organ failures have a particularly high associated mortality. Liver failure in the context of multi-organ failure (MOF) is a poor sign, so hypoglycaemia with high bilirubin and lactate is worrying. The presence of disseminated intra-vascular coagulation (DIC) is similarly ominous, and a simple DIC score using INR, platelets, fibrinogen and D-dimers has good predictive accuracy for mortality. The response to therapy is also important, and any failure to improve or deterioration after support has started is a poor sign. The response to treatment may be tracked sequentially using SOFA scores.

How long would you continue organ support?

Active treatment should continue for as long as the risks and burdens were outweighed by the benefits, i.e., the possibility of survival with a quality of life meaningful to the patient. Each decision is made on an individual basis considering the clinical context and the wishes of the patient if they are known or can be ascertained. The relatives can give useful information bearing on the patient's best interests and their wishes should also be taken into consideration. It remains a medical decision, although the recent Mental Capacity Act contains guidance on how to proceed when considering the best interests of incompetent patients.

What are the long-term effects on survivors?

Long-term effects can be considered in two groups: those that are present in hospital and those that persist after discharge home. Some sequelae of MOF are slow to resolve and lead to problems within hospital and indeed delay discharge home. In particular, critical illness polyneuropathy and polymyopathy are associated with MOF and perhaps could be considered part of neurological system failure. Muscle weakness is the most obvious manifestation and leads to prolonged dependency because of poor mobility and muscle strength. Even feeding oneself may be difficult or impossible and nutritional supplements or nasogastric/jejunal feeding may be needed for some time.

Is there anything else?

Many things that we take for granted, such as sleep or bowel and bladder function, may take time to return to normal after MOF. Delirium is another neurological failure that can be very subtle and slow to resolve, and may be mis-attributed to memory impairment or considered a normal response to the events surrounding critical illness. For some, particularly the elderly, any of these effects may prevent a return to home or independence.

Tell me about mortality and morbidity after discharge

Overall mortality is increased after discharge from hospital. This is affected by age and co-morbidity, but there is a clear effect of MOF that lasts for a year or more. Of those who survive, between 25 and 50% need assistance with activities of daily living at 1 year, although it is much less than this at 3 years; again this is affected by age and previous functional status. Few will require long-term respiratory support, and less than 10% develop the need for long-term renal support for example, but testing will reveal sub-clinical deficits. After ARDS a restrictive deficit with reduced diffusing capacity is present for months. Studies of cognitive function suggest that problems persist for months or years afterwards across several domains such as attention, memory, task planning and execution.

What are the social implications for these patients? And how do they feel about it?

This can cause problems with return to work, relationships and general quality of life. Using validated questionnaires, most patients report a lower quality of life than matched controls, but most would undergo the ICU episode again.

Are there any other problems that arise from a long-term ICU stay? How can patients with problems be identified and treated?

There are many other problems that can result, but there are insufficient data to make confident estimates of the size of the problem in the UK. Chronic pain, chronic fatigue and sleep problems occur, as does anxiety, depression and post-traumatic stress disorder. These may be difficult to diagnose or may not be attributed to time spent in ICU with multi-organ failure. As attention turns from simple mortality based statistics to patient related outcome measures such problems are likely to be better identified, and recent NICE guidance emphasises the importance of multidisciplinary rehabilitation after critical illness. An ICU follow-up clinic is one way in which patients can be identified earlier and appropriate support and treatment organised.

Why might scoring systems be used in ICU?

Scoring systems enable a representation of disease severity as well as an estimation of in-hospital mortality which can be utilised in a number of ways in ICU. They can assist in decision making for clinicians, allow comparison of outcomes for different Intensive Care units and facilitate discussions about severity of illness with family members of patients. They may also be used for research purposes, allow auditing of unit practice and if calculated regularly can monitor response to treatment.

Typically scoring systems consist of a dimensionless number relating to disease severity with increasing numbers indicating more severe disease alongside a probability of mortality. Overarching categories of scoring system include disease-specific and generic with a subset of common scores in day-to-day use being physiological scores. These encompass illness severity scores (such as the Sequential Organ Failure Assessment (SOFA) score), models for outcome prediction (such as the APACHE II score) and decision support tools which include the various iterations of the early warning score system.

What constitutes an effective scoring system?

Scoring systems should be simple and easy to use and interpret. They should be valid and be developed in a population similar to the one that they are being applied to. They should show discrimination and the metric used for this is the area under the receiver operator curve (AUROC) produced by comparing specificity and sensitivity. The closer the AUROC is to 1 the better the test performs. Finally, they should be well calibrated and show good uniformity of fit which allows them to be used for different subgroups of patients rather than just the general ICU population.

Which scoring systems do you know of? Can you explain the use of them?

Scoring systems can initially be categorised into disease-specific scores and generic scoring systems. Disease-specific scores include commonly used scores such as the Glasgow Coma Score and the UK Model for End-Stage Liver Disease (UKELD). These are used to predict a level of disease severity based on various physiological parameters relevant to the disease being assessed and each will have their own scale of what cumulative score constitutes different levels of severity. Other examples include the Kidney Disease Improving Global Outcomes (KDIGO) score for acute kidney injury, Ranson's criteria for pancreatitis and the Rockall score for upper gastrointestinal bleeding.

Generic scoring systems can be categorised into outcome prediction models, illness severity scores and therapeutic weighting scores. Outcome prediction models include the Acute Physiology and Chronic Health Evaluation (APACHE) score and the Simplified Acute Physiology Score (SAPS) amongst others. APACHE is perhaps the most widely used and takes physiological variables within the first 24 hours of admission to ICU, age and markers of organ system to produce a score that allows prediction of in-hospital mortality.

Generic illness severity scores include the SOFA and Multiple Organ Dysfunction Score (MODS) amongst others. The SOFA score is widely used on the premise that acute illness produces related organ dysfunction and various organ-based physiological parameters in six domains are combined to produce an overall score. While this does not predict mortality, high scores are associated with increased mortality.

The most common therapeutic weighting scale is the Therapeutic Intervention Scoring System used to quantify nursing workload. It follows that more unwell patients need a greater number and higher acuity of interventions and seven groups of organ-based nursing interventions are combined to assess severity of illness and compare patient care.

Further Reading

Bouch D, Thompson J. Severity scoring systems in the critically ill. *British Journal of Anaesthesia* 2008; 8(5): 181–185.

Department for Constitutional Affairs (DCA). Mental Capacity Act 2005 – Summary. <http://webarchive.nationalarchives.gov.uk/+/www.dh.gov.uk/prodconsumdh/groups/dhdigitalassets/@dh/@en/documents/digitalasset/dh4108596.pdf>.

Desai N, Gross J. Scoring systems in the critically ill: Uses, cautions, and future directions. *British Journal of Anaesthesia*. 2019; 19(7): 212–218.

Hofhuis J, van Stel H, Schrijvers A, et al. Health related quality of life in critically ill patients. *Current Opinion in Critical Care*. 2009; 15: 425–430.

National Institute for Health and Clinical Excellence (NICE). Rehabilitation after critical illness. 2009. <http://www.nice.org.uk/nicemedia/live/12137/43526/43526.pdf>.

Singer M, De Santis V, Vitale D, Jeffcoate W. Multiorgan failure is an adaptive endocrine-mediated metabolic response to overwhelming systemic inflammation. *Lancet*. 2004; **364**: 545–548.

Strand K, Flaatten H. Severity scoring in the ICU: a review. *Acta Anaesthesiologica Scandinavica*. 2008; **52**: 467–478.

Vincent JL, Moreno R, Takala J. The SOFA (Sepsis-related Organ Failure Assessment) score to describe organ dysfunction/failure. On behalf of the Working Group on Sepsis-Related Problems of the European Society of Intensive Care Medicine. *Intensive Care Medicine*. 1996; **22**: 707–110.

3.2.3 ARDS and Ventilation Difficulties – Matt Thomas

The management of ARDS is a core topic in intensive care, and will also be relevant to all who will have to anaesthetise high-risk and critically ill patients. In the structured oral examinations (SOEs) it may be a clinical scenario or follow a discussion of respiratory physiology, for example, the different causes of hypoxia.

What are the causes of hypoxia? Please give me clinical examples of each.

Even if you are not asked to define a particular term it is worth stating briefly what you understand by it so that you (a) demonstrate knowledge to the examiners and (b) allow any possible misunderstanding of the question to be clarified before you have gone too far.

Hypoxia is an inadequate tissue oxygen supply or use and has four main causes:

- Hypoxic
- Anaemic
- Ischaemic/stagnant
- Cytopathic/histotoxic.

Hypoxic hypoxia is a reduction of oxygen uptake by the lung, for example after pneumonia. Anaemic hypoxia results from reduced arterial oxygen content secondary to loss of haemoglobin, or presence of abnormal haemoglobins; examples are major haemorrhage and carbon monoxide poisoning respectively. Ischaemic hypoxia results from a reduced oxygen delivery secondary to reduced cardiac output or local vascular occlusion, for example cardiogenic shock or arterial thromboembolism. Cytopathic hypoxia results from disrupted cellular metabolism, for example cyanide poisoning of mitochondrial respiratory chain enzymes. Different types may coexist.

What are the causes of hypoxic hypoxia?

There are several causes:

- Reduced inspired oxygen concentration (FiO_2)
- Hypoventilation
- Ventilation/perfusion (V/Q) mismatch
- Shunt
- Shunt with venous admixture
- Reduced diffusing capacity.

Tell me a little more about V/Q mismatch.

The alveolus is the functional unit of gas exchange. It is where the gas breathed in to the lung (ventilation) meets the blood pumped through the lung (perfusion). If we assume that the cardiac output and inspired oxygen concentration (FiO_2) are in a steady state

then the PaO_2 is determined by the balance between ventilation and perfusion across all alveoli. A disturbance of the normal balance is called ventilation-perfusion or V/Q mismatch and leads to impaired gas exchange. It is the most common cause of hypoxia.

Can you explain how hypoxia may arise from V/Q mismatching?

In a healthy lung mechanisms such as hypoxic pulmonary vasoconstriction work to keep ventilation and perfusion balanced so gas exchange is as efficient as possible. No ventilation or perfusion is wasted. As there is deviation from a 1:1 ratio whether this is a change in V relative to Q or in Q relative to V gas exchange is less efficient and overall oxygen transfer will be reduced. Some variation around the perfect 1:1 ratio throughout the lung is normal but usually minimal. As the spread increases as a result of disease the effect on gas exchange also increases.

How is this different from a shunt?

In cases of true shunt, alveoli are ventilated but not perfused, so no oxygen can be taken up into the pulmonary venous blood. This may be anatomical, like an arteriovenous malformation (AVM), or physiological such as atelectasis or pneumonia. As the overall amount of shunt increases, the shunt fraction, the total oxygen content of pulmonary venous blood will fall. Alveoli that are ventilated will usually fully saturate the blood leaving them so cannot compensate by transferring more oxygen. That means 100% oxygen does not correct the hypoxia seen with shunting. I should say that shunt can also be extrapulmonary, that is cardiac, and that the right-to-left variety have the same effect of deoxygenated blood bypassing the lung.

What is the significance of diffusion impairment?

In most pathological conditions gas exchange is not diffusion limited since the alveolar-capillary barrier is very thin and so highly gas permeable. Equilibration between alveolar gas and pulmonary capillary blood is normally very fast, normally one-third of the contact time of 0.75 seconds in the capillary. However, if the alveolar membrane is thickened, like in ARDS, or capillary transit times are quick (high cardiac output states), then this may contribute to hypoxia.

How about hypoxia from hypoventilation?

With hypoventilation alveolar O_2 falls and CO_2 rises so that there is less oxygen available to transfer to the blood. Note that this is also important if dead-space volume approaches tidal volume. If the dead-space increases then a greater portion of the tidal volume does not reach the alveoli and participate in gas exchange. The effective alveolar minute ventilation is reduced, and this means that alveolar CO_2 rises and also, in poorly ventilated alveoli, V/Q ratio falls. Overall alveolar oxygen content falls and therefore so does pulmonary oxygen uptake.

Are you aware of any clinical conditions in which dead-space fraction is related to mortality?

The two clinical conditions where this has been demonstrated are pulmonary embolism and ARDS.

What is the current definition of ARDS?

The definition is the Berlin definition of 2012. It has four components: timing, chest imaging, origin of oedema and oxygenation. Essentially it needs to be acute, less than a week, with bilateral opacities on either chest X-ray or CT that are not fully explained by effusions, nodules or lobar disease. It cannot be simply cardiac failure or fluid overload although of course they may coexist. Lastly the $\text{PaO}_2 : \text{FiO}_2$ ratio must be less than 40 kPa with PEEP or CPAP of at least 5 cm of water.

How do you grade severity?

By $\text{PaO}_2 : \text{FiO}_2$ ratio. Less than 13.3 is severe, between 13.3 and 26.6 is moderate and between 26.6 and 40 is mild. Strictly speaking only intubated patients can have moderate or severe ARDS as only PEEP and not CPAP is permitted by the definition.

What are the causes of ARDS?

It is important to remember that ARDS is not a disease but a syndrome with an underlying cause. Actually there are many, conveniently considered in two categories: pulmonary and extra-pulmonary.

Pulmonary causes result from direct insults to the lung and include:

- Pneumonia aspiration smoke inhalation
- Pulmonary contusion.

Extra-pulmonary or indirect causes include:

- Sepsis
- Severe trauma transfusion reactions burns
- Acute pancreatitis drug reactions.

There are also conditions that may mimic ARDS such as connective tissue disease or vasculitides. The key is in the clinical history although there can be typical patterns on chest CT scan, bronchoalveolar lavage cytology or autoimmune profiling.

Does this classification help?

There is a lot of debate about whether or not pulmonary and extra-pulmonary ARDS are different diseases, and I don't think this has been resolved. For practical purposes most treat them in the same way. Ventilator support is titrated to individual lung mechanics rather than on the basis of the trigger for lung failure.

Tell me about the pathophysiology of ARDS?

There are two stages that may be distinguished histologically: the exudative phase followed by the fibroproliferative phase. First there is an acute inflammatory reaction in the lung, and the alveoli fill with neutrophils, blood and a protein-rich pulmonary oedema. There is associated alveolar epithelial and capillary endothelial damage, with loss of alveolar cells and structural integrity and capillary thrombosis. Days or weeks later, chronic inflammation, fibrosis and neovascularisation occur. Reduction of inflammation and prevention of progression to fibrosis are the basis of the rationale for steroids.

We'll come back to that later. For now, tell me how does ARDS cause hypoxia? Can you relate this to our earlier discussion?

There is widespread and profound V/Q mismatching as a result of the inflammatory reaction, which may be superimposed on a direct lung injury. Both shunt and dead-space increase, as alveoli and capillaries respectively are occluded. Oedema and later fibrosis affect diffusion. If severe, right ventricular failure and low cardiac output contribute further to venous admixture and hypoxia.

OK, now I would like you to consider this scenario.

You are asked to review a previously fit 25-year-old man admitted to hospital after a motorcycle crash. He is in recovery following a femoral nailing under general anaesthesia, during which he had two red cells and two FFP transfused. His respiratory rate is 30 breaths/min and has an oxygen saturation (SpO_2) of 89% on 15 l of O_2 via a reservoir mask. He is agitated with obvious respiratory distress and a sinus tachycardia; further examination is essentially unremarkable. What could be the problem?

Some opening statements might seem too obvious, but do serve to tell the examiner that you are able to recognise a critically ill patient promptly and appreciate situations calling for urgent action. If asked for a differential diagnosis in these scenarios, start with the most likely given the history.

This young man is clearly unwell, given his high respiratory rate and low SpO_2 both of which are out of keeping with the expected clinical course. It is possible that post-operative respiratory distress could arise from incomplete reversal of neuromuscular blockade and this should be excluded using a peripheral nerve stimulator. He could also have severe pain, although the hypoxia does not quite fit. More concerning are other potential causes of acute respiratory distress which in this situation include aspiration, fat embolism or a transfusion-related-acute-lung-injury.

How will you proceed?

I would begin with an arterial blood gas, a 12-lead ECG and a chest X-ray. I would also review the admission imaging to see if there is anything that could have deteriorated intraoperatively, for example pulmonary contusions or intra-abdominal injury. As he will need respiratory support and monitoring beyond the capability of the ward to provide, I would refer to ICU.

What kind of respiratory support do you mean?

In the first instance high flow oxygen should be used and non-invasive ventilation, particularly CPAP, can be used for acute hypoxaemic respiratory failure. In this case, with his rapid deterioration and agitation he is likely to require mechanical ventilation.

How are you going to set up the ventilator?

If you are going to quote any trials to support what you say then you must have a clear idea of what the trial showed and any major criticisms, though a full critical appraisal is not required.

I will assume that this problem is not a tension pneumothorax or lobar or lung collapse but ARDS related in some way to his major trauma.

A reasonable starting point is the lung-protective strategy used in the ARDSnet trial. I would use pressure-controlled ventilation to aim for a tidal volume of 6 ml/kg of ideal

body weight, providing plateau pressures are less than 30 cm of water. I would start with PEEP at 10–12 cm H₂O and titrate the FiO₂ aiming for oxygen saturations of 88–95%. I would worry about CO₂ once there is evidence of severe respiratory acidaemia, a pH under 7.25. The options include increasing tidal volume or respiratory rate and the choice will depend on lung compliance and driving pressure.

Did the ARDSnet trial use pressure-controlled ventilation?

Volume-controlled ventilation was used in the trial. Many believe pressure-controlled ventilation allows better control of plateau pressures and has gas exchange benefits. Delivering lung-protective ventilation and titrate ventilator settings to the patient is probably more important than the mode you start with.

Let's talk about titration in that case. You mentioned options for CO₂ control, please elaborate.

Driving pressure, or the difference between PEEP and plateau pressure, is increasingly recognised as important. It reflects lung compliance and although no prospective trials exist evidence suggests a threshold for harm at 15 cm of water. If driving pressure is below 15 cm of water there is scope to increase tidal volume by increasing driving pressure. If above 15 cm water then ideally tidal volume would be reduced and respiratory rate increased to increase minute ventilation and control CO₂. Again, in the absence of brain injury, CO₂ does not need to be normal.

And how do you titrate the PEEP?

Setting optimal PEEP is still much discussed. Too high and there are harmful effects on cardiac output and lung overdistension, too low and there is persistent hypoxia and atelectasis. There are many ways described but a simple method is to titrate PEEP to the best – that is lowest – driving pressure for a fixed tidal volume and to observe the effects on haemodynamics, oxygenation and dead-space (using CO₂ as a surrogate if minute ventilation unaltered).

What are the principles of management of ARDS in the ICU?

There is specific and supportive care. Specific care includes treating the cause of ARDS and limiting further lung damage with a lung-protective ventilatory strategy. There is interest in the concept of guiding lung protection settings by minimising the mechanical power, crudely estimated as 4x driving pressure (DP) plus respiratory rate (RR) [(4 × DP)+RR], to reduce the stress and strain forces that damage the lung during ventilation. Fluid balance is important as the ARDSnet fluids and catheters study suggests that keeping the lung dry is associated with better outcomes. Supportive care for ARDS is as for any critically ill patient, for example VTE prophylaxis, minimising sedation, enteral nutrition, infection control.

What does a lung-protective ventilation strategy aim to avoid?

It is a ventilation strategy that is based on the premise that ventilation causes injury to the sick lung in a number of ways. The major causes of injury are volutrauma, barotrauma, atelectrauma and biotrauma.

What do these terms mean?

In ARDS not all the lung is equally involved in the pathological process. Essentially excessive tidal volume and inflation pressure over-distend healthy alveoli, hence volu-

and barotrauma, and the diseased alveoli are subjected to cyclical opening and closing, hence atelectrauma. These concepts are evolving into the more precise but less intuitive ones of stress and strain describing the ventilator derived forces acting on the lung that can damage tissue. The damage to the alveolar–capillary barrier leads to increased permeability, release of inflammatory mediators and translocation of pathogens, hence biotrauma.

Are there specific treatments for ARDS itself?

As yet there is no proven therapy for established ARDS, although not for want of trying.

Is there a role for steroid therapy?

There is still controversy regarding steroids in ARDS, even allowing for the success of dexamethasone in COVID-19 as large multicentre prospective trials in non-COVID ARDS are still lacking. However, if given at moderate doses within the few first days and tapered slowly there appear to be benefits in gas exchange and reduced time on the ventilator. Given later, any benefit is outweighed by the risks of neuromuscular problems, immunosuppression and increased mortality. The ideal steroid, dose and duration is not known. Nor are the patients who should have steroids: ARDS is a heterogenous syndrome and at least two subphenotypes exist that differ in response to statins and fluid for example. It may be that steroid response also differs.

Despite optimal management of this patient, his gas exchange continues to deteriorate. What are your options?

The initial diagnosis might need to be reconsidered and complications such as pneumothorax, pneumonia or pulmonary embolus should be excluded. A chest CT scan is helpful both for this and to give an idea of the pattern of lung involvement to guide selection of rescue therapies. It is easy to miss ‘fluid creep’ and attention should be paid to fluid balance. A cardiac echo can exclude cardiac injury and quantify right heart function.

The patient’s oxygenation continues to deteriorate: What will you do now?

The first line thing I would do is paralyse the patient. Then the next rescue strategy I would use is prone ventilation which does have a mortality benefit in severe ARDS. This is best considered at an early stage and maintained for long periods, up to 18 to 20 hours at a time.

What problems are associated with prone ventilation?

The main problems relate to the process of turning and the prone position itself. Turning safely requires numerous staff but with familiarity and practice can be done for the majority of patients.

The most common manoeuvre-related complications are:

- Airway obstruction or accidental extubation
- Transient hypoxia
- Hypotension and arrhythmias
- Vomiting
- Accidental loss of drains or lines.

Nursing in the prone position can be difficult (suction/mouth care, etc.) and practical procedures such as urinary catheterisation, insertion of central, peripheral and arterial lines can be impossible. Patients often develop facial oedema. Care must be taken to prevent ocular, joint and peripheral nerve injuries. Despite everything pressure damage to skin is common.

What other methods can be considered to improve oxygenation?

Pulmonary vasodilators such as nitric oxide or inhaled prostacyclin can improve blood gases and might help with right ventricular failure though the effect on mortality is debated. Airway pressure release ventilation can improve lung recruitment and oxygenation but might not suit haemodynamically unstable patients or those with airway obstruction. Again there is little solid evidence of mortality benefit. Lastly there is referral for Extracorporeal Membrane Oxygenation (ECMO) if there is refractory severe acute respiratory failure.

What is the mortality associated with ARDS?

In recent randomised controlled trials the mortality has been around 25–30%, but community surveys show mortality of about 35–40%. This may reflect the difficulty of translating research findings into clinical practice.

What are the predictors of outcome?

The age of the patient and the number of non-pulmonary organ failures are the best predictors. Elderly patients with shock do badly, as do those with hepatic failure or interstitial lung disease. Surprisingly the initial $\text{PaO}_2 : \text{FiO}_2$ ratio is not predictive, unless it is under 50 or fails to improve during the first week.

What is the mode of death in ARDS?

Death is usually due to other organ failures and sepsis. It is rarely intractable severe hypoxia itself.

What is function like in survivors of ARDS?

There are persistent problems, the post-intensive care syndrome, in many survivors. Lung function deficits are present in most, although this is rarely a problem in day-to-day life and return to nearly normal within the first year. Cognitive, psychological and neuromuscular complications can be frequent and severe and more likely to prevent return to work or precipitate loss of independence. Survivors do have a reduced health-related quality of life.

Further Reading

Bos LDJ, Ware LB. Acute respiratory distress syndrome: Causes, pathophysiology, and phenotypes. *Lancet*. 2022; 400: 1145–1156.

Ferguson N, Fan E, Camporota L et al. The Berlin definition of ARDS: An expanded

rationale, justification and supplementary material. *Intensive Care Medicine*. 2012; 38: 1573–1582.

Gattinoni L, Tonetti T, Quintel M. Regional physiology of ARDS. *Critical Care*. 2017; 21 (Suppl 3): 312.

Guidelines on the management of the Acute Respiratory Distress Syndrome Version 1 (2018). The Faculty of Intensive Care Medicine and The Intensive Care Society. https://ficm.ac.uk/sites/ficm/files/documents/2021-10/Guidelines_on_the_Management_of_Acute_Respiratory_Distress_Syndrome.pdf accessed 28 August 2022.

Peliso P, Ball L, Barbas C et al. Personalized mechanical ventilation in acute respiratory

distress syndrome. *Critical Care*. 2021; 25: 250.

Petersson J, Glenny R. Gas exchange and ventilation-perfusion relationships in the lung. *European Respiratory Journal*. 2014; 44: 1023–1041.

Reilly J, Calfee C, Christie J. Acute respiratory distress syndrome phenotypes. *Seminars in Respiratory and Critical Care Medicine*. 2019; 40: 19–30.

3.2.4 The Management of Sepsis-Daniel El – Dalil, Charlotte Morris and Gareth J Gibbon

You are most likely to have to confront this topic as part of a case-based discussion. You are asked to urgently review a 47-year-old man in the resuscitation area of the emergency department who is in respiratory distress and hypotensive.

How would you approach this patient?

The examiner is looking for a safe, systematic approach to managing an unwell patient. It is sensible to have a generic answer to start every similar question – this gives you time to think and makes sure you don't miss out on any easy marks.

I would approach this patient as I would every potential medical emergency. I would assess the airway, breathing and circulation, apply high flow oxygen via a hudson mask with a reservoir bag and, if concerned, would call for help early. I would ensure large bore intravenous access is secured and, providing there is no obvious contraindication, I would give a bolus of intravenous fluids. I would set up basic monitoring: a pulse oximeter, frequent non-invasive blood pressure measurements and, if available, ECG telemetry. When safe to do so I would attempt to make a diagnosis through a history, examination and by requesting relevant investigations. These would include blood investigations including: a full blood count, urea and electrolytes, clotting, two sets of blood cultures, an arterial blood gas including a lactate level. As well as this I would like a 12-lead ECG, a rapid COVID swab and a chest X-ray.

What would you like to know about him?

It is always sensible to start from the beginning. It gives you time to formulate your thoughts. It also may help to imagine yourself in that situation.

I would like to start by doing an A–E assessment as he is an unwell gentleman and then follow this by taking by a history. My first concern is his airway. Is he able to talk to me? **Yes, but he is struggling to complete sentences.**

He is already receiving high flow oxygen via a non-rebreathe mask. I would like to know his respiratory rate and his oxygen saturation? I would continue to think about his breathing and listen to his chest. I would ask for a portable chest X-ray to be ordered if this had not already been done.

You can hear coarse crepitations over the right side of his chest, but his left chest sounds clear. His oxygen saturations are 93% on the mask you have put on and his respiration rate is 40 breaths per minute.

I would like to proceed to assess his circulation. I would like to know his heart rate, blood pressure, and central capillary refill time? I would ask for a 12-lead ECG if it had not already been performed. Has he had an arterial blood gas sampled? Are there any blood results?

His blood pressure is 74/45 mmHg, he has a capillary refill of 4 seconds, his ECG shows a sinus tachycardia, his blood gases show a metabolic acidosis with a lactate of 4.4mmol/l, and his chest X-ray shows right lower lobe shadowing. He has a temperature of 39°C and feels very flushed.

I would then like to take a brief history enquiring how long he had been unwell, his main symptoms, if he has any associated chest pain and if he has had any weight loss, his past medical history and drug history and allergy status, and when he last had anything to eat or drink.

He is able to tell you that he has been unwell for the past 48 hours with a worsening cough, fevers, and breathlessness. He has had no weight loss. He has no other significant medical history, takes no medicines regularly and has no known allergies. He last ate 6 hours ago.

What are your thoughts?

Remember – you might also be given a copy of the investigations and asked to describe them. Ensure you have a systematic way of doing so. Usually, you will see the relevant investigations before the SOE.

This is an unwell middle-aged gentleman with right lower lobe air space shadowing on his chest X-ray and my top differential diagnosis would be septic shock from a right lower lobar pneumonia.

As this gentleman is very unwell, I would be wanting to perform an HIV screen to see if he has impaired immunity predisposing to severe infection. As well as this I would perform an atypical pneumonia screen, extended viral screen and consider sputum sampling for tuberculosis depending on his X-ray and risk factors.

Are you able to define sepsis, how would you further classify sepsis and how is it diagnosed?

Sepsis is defined as dysregulated host response to infection causing life-threatening organ dysfunction. These include hypoxaemia, oliguria, a significant increase in creatinine, a coagulopathy, thrombocytopenia, ileus, or hyperbilirubinemia.

Septic shock is a subset of sepsis in which there is circulatory failure and a higher associated mortality. It can be defined as sepsis with hypotension (I would define this as a MAP <65 mmHg) or a raised lactate (>2 mmol/L) despite adequate fluid resuscitation.

Sepsis was previously defined as systemic inflammatory response syndrome with suspected underlying infection. Current definitions have moved away from this as the SIRS criteria lacked specificity due to many hospitalised patients without sepsis meeting the SIRS criteria. Despite this SIRS is still recommended as a screening tool for sepsis. Other scores such as SOFA and qSOFA have been developed to predict mortality of patients with sepsis.

The Surviving Sepsis Campaign has specifically advised against using qSOFA in isolation as a screening tool for sepsis as it underperformed compared to other screening methods such as the National Early Warning Score or Modified Early Warning Score or SIRS.

How would you manage this patient?

Having commenced fluid resuscitation and administered oxygen I would ensure two sets of blood cultures were taken and broad-spectrum antibiotics were administered as soon as possible. This is most likely to be a community acquired pneumonia so you would need to cover pneumococcus, haemophilus, and atypical organisms. In my hospital we would administer intravenous co-amoxiclav and clarithromycin.

As previously stated, this patient is in septic shock, he has a lactate of 4.4 mmol/L which suggests significant tissue hypoperfusion and puts him in a high-risk group. He will require Critical Care support for advanced monitoring, vasopressors, and likely invasive ventilation as he is in type 1 respiratory failure with a very high respiratory rate. I would arrange for safe transfer from his current location to a critical care unit with resuscitation ongoing.

What else would you do for him?

With a working diagnosis of septic shock, I would like to insert an arterial line so that his mean arterial pressure can be continuously monitored as well as facilitate easy arterial blood gas monitoring. I would be aiming for a mean arterial pressure (MAP) target of >65 mmHg.

I would also like to insert a central line as this will allow us to administer vasopressors should his blood pressure not respond to adequate fluid resuscitation. I would also insert a urinary catheter so that this patient's urine output could be closely monitored. To assess the patient's fluid status, I would be monitoring the patient's capillary refill time, urine output, lactate levels, as well as his response to fluids.

If the patient remained hypoxic with a respiratory rate of greater than 40, I would strongly consider whether this patient needs invasive ventilation.

I would also ensure the patient received prophylactic low molecular weight heparin, stress ulcer prophylaxis as well as monitoring his blood glucose levels and initiating insulin therapy if needed to keep them <10 mmol/L.

What would you do if the patient remained hypotensive despite fluid resuscitation?

If I was confident in my diagnosis of vasodilatory shock then I would start a noradrenaline infusion centrally titrated to response, running up to $0.5 \mu\text{g/kg/min}$. If the hypotension proved refractory to higher doses of vasopressors, then low-dose steroids should be considered. Once the patient was requiring doses of noradrenaline of $0.5 \mu\text{g/kg/min}$ I would strongly consider the addition of vasopressin as a second line agent to manage the refractory shock.

You have mentioned using steroids, can you tell me about any recent papers on steroids in sepsis?

The most recent and largest study investigating steroids in sepsis was the ADRENAL trial from 2018; this was a multinational, multicentre double-blinded randomised control trial, investigating whether a hydrocortisone infusion had 90-day mortality benefit over placebo in patients with septic shock. This demonstrated no statistically significant difference in mortality but there was significant reduction in time taken for shock to resolve, time to discharge from ITU, initial number of days of mechanical ventilation

and use of blood transfusion. The most recent 2021 surviving sepsis guideline suggests using IV corticosteroids in patients with ongoing vasopressor requirements.

What do you know about early goal directed therapy?

Early goal directed therapy refers to a bundle of care which focussed on intensive monitoring of circulatory parameters and aggressive management of these with the aim of optimising tissue perfusion. These parameters included:

- CVP 8–12 mmHg
- MAP 65–90 mmHg
- Urine output 0.5 ml/kg/hr
- Central venous oxygen saturation >70%
- Haematocrit >30%

This was initially recommended following a RCT by Rivers; however, subsequent studies: ProCESS, ARISE and ProMISE have all demonstrated no benefit for aiming for early goal directed therapy over standard care. In place of the strict protocol of early goal directed therapy the fundamentals of high-quality management of patients with sepsis are early recognition, prompt antibiotic initiation, source control, fluid resuscitation and escalation to higher level care if appropriate.

Further Reading

- Evans L, Rhodes A, Alhazzani W, Antonelli M, Coopersmith CM, French C, Levy M. Surviving sepsis campaign: International guidelines for management of sepsis and septic shock 2021. *Intensive Care Medicine*. 2021; 47(11): 1181–1247.
- Mouncey PR, Osborn TM, Power GS, Harrison DA, Sadique MZ, Grieve RD, Rowan KM. Trial of early, goal-directed resuscitation for septic shock. *New England Journal of Medicine*. 2015; 372(14): 1301–1311.
- Peake SL, Bailey M, Bellomo R, Cameron PA, Cross A, Delaney A, Australian and New Zealand Intensive Care Society Clinical Trials Group. Australasian resuscitation of sepsis evaluation (ARISE): A multi-centre, prospective, inception cohort study. *Resuscitation*. 2009; 80(7): 811–818.
- ProCESS Investigators. A randomized trial of protocol-based care for early septic shock. *New England Journal of Medicine*. 2014; 370(18): 1683–1693.
- Rivers E, Nguyen B, Havstad S, Ressler J, Muzzin A, Knoblich B, Tomlanovich M. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *New England Journal of Medicine*. 2001; 345(19): 1368–1377.
- Singer M, Deutschman CS, Seymour CW, Shankar-Hari M, Annane D, Bauer M, Angus DC. The third international consensus definitions for sepsis and septic shock (Sepsis-3). *Journal of the American Medical Association*. 2016; 315(8): 801–810.
- Venkatesh B, Finfer S, Cohen J, Rajbhandari D, Arabi Y, Bellomo R, Myburgh J. Adjunctive glucocorticoid therapy in patients with septic shock. *New England Journal of Medicine*. 2018; 378(9): 797–808.

3.2.5 Oxygen Toxicity – Will H Spencer, Emily K Johnson and Jessie R Welbourne

As anaesthetists, our most used drug is oxygen. However, its use is not without problems. Therefore, you may have an MCQ or structured oral examination (SOE) asking about the adverse effects of oxygen therapy or more specifically about oxygen toxicity.

What are the issues associated with giving too much oxygen?

Take your time and gather your thoughts. With a broad question like this it is vital you classify your answer. Don't rush into an answer about free radicals you may later regret! If the examiner wants to know about the mechanisms of oxygen toxicity, they will lead you down this path of questioning in due course. It is important to show a logical approach and mention the common problems first.

The problems with excessive oxygen therapy can be divided into body systems:

Pulmonary:

- Pulmonary oxygen toxicity
- Hypercapnic respiratory failure
- Absorption atelectasis
- Bronchopulmonary dysplasia (neonates)
- Bleomycin-induced lung injury

Neurological:

- CNS toxicity, particularly in hyperbaric conditions (eg diving)
- Cerebral vasoconstriction

Ocular:

- Retinopathy (retinopathy of prematurity in neonates)

Cardiovascular:

- Coronary vasoconstriction
- Potential increase in infarct size in STEMI (AVOID trial).

What are the mechanisms of oxygen toxicity?

Reactive oxygen species (ROS, also known as free radicals) are the likely aetiological factor in oxygen toxicity. These are highly reactive oxygen molecules with unpaired electrons. They are created through various molecular processes including:

- Mitochondrial oxidative processes
- Xanthine oxidase at extramitochondrial sites
- Auto-oxidative reactions
- Phagocytosis of bacteria

ROS include the superoxide anion (O_2^-), the singlet oxygen ($^1\text{O}_2$) and the highly toxic hydroxyl radical ($\text{OH}\cdot$). ROS exert numerous harmful effects, including:

- Damage to DNA and RNA
- Impairment of DNA repair mechanisms
- Disruption to transcription
- Disruption to protein synthesis
- Inactivation of cellular enzymes.

Under normal conditions, antioxidants such as glutathione peroxidase, catalase and superoxide dismutase deactivate ROS. However, in hyperoxic conditions, the excess production of ROS overwhelms antioxidant capacity, leading to cellular damage.

Describe the pulmonary effects of oxygen toxicity.

Pulmonary toxicity can occur due to prolonged exposure to high concentrations of oxygen causing damage to the pulmonary epithelium and inactivation of surfactant. This can lead to alveolar oedema, increased secretions and interstitial thickening which can progress to fibrosis.

4 stages have been described (Horncastle, 2019):

- Initiation: hyperoxia causes a mismatch between ROS and antioxidants.
- Inflammation: migration of inflammatory mediators, damage to pulmonary epithelium, oedema and hyperpermeability.
- Proliferation: increased presence of inflammatory cells, increased secretions, and cellular hypertrophy.
- Fibrosis: collagen deposition and thickening of the interstitium. This stage is predominantly irreversible.

Furthermore, bronchopulmonary dysplasia can occur in infants exposed to high concentrations of oxygen.

Symptoms and signs include:

- Tracheal irritation
- Cough
- Chest pain
- Pulmonary oedema
- Reduction in vital capacity (one of the earliest measurable signs).

You mentioned bronchopulmonary dysplasia, can you tell me more?

Bronchopulmonary dysplasia (BPD) is a form of chronic lung disease that predominantly affects premature infants, particularly those exposed to supplemental oxygen and mechanical ventilation. It is defined as a neonate with respiratory distress who requires >21% oxygen for at least 28 days. The incidence rises sharply with degree of prematurity. Mechanical stress, oxygen toxicity, and premature lung development all contribute to its severity. Adverse sequelae include impaired respiratory function, increased susceptibility to infection, reactive airway disease, pulmonary hypertension, and pulmonary fibrosis. The introduction of treatments such as prenatal steroids, postnatal surfactant, lung-protective ventilation, and improved nutrition have improved its prognosis.

Tell me about absorption atelectasis.

This is the process by which alveoli collapse due to the rapid absorption of oxygen. It occurs due to the high solubility of oxygen compared with nitrogen. When breathing air, the relative insolubility of nitrogen allows terminal airways and alveoli to be 'splinted' open. When nitrogen is replaced with oxygen, the gas in the alveoli is more readily absorbed leading to airway collapse and loss of alveolar volume.

Why is it dangerous to give excess oxygen to patients with COPD and a chronically raised PaCO₂?

Excess oxygen in this group can lead to hypercapnic respiratory failure. This occurs as a result of:

1. V/Q mismatch – excessive oxygen reverses hypoxic pulmonary vasoconstriction causing increased perfusion to diseased areas of the lung. This leads to shunt, an inability to clear CO_2 , and hypercapnia.
2. The Haldane effect: deoxygenated haemoglobin has a greater affinity for CO_2 than oxygenated haemoglobin. By increasing the partial pressure of oxygen in the blood, CO_2 is displaced from haemoglobin and PaCO_2 increases.

The combination of these effects is the current model for understanding the dangers of excessive oxygen therapy in patients with chronically raised PaCO_2 , rather than the 'hypoxic respiratory drive' concept.

Describe the effects of oxygen toxicity on the central nervous system.

Central nervous system toxicity is a concern to those encountering oxygen at a high barometric pressure, such as divers. It is due mainly to oxidation and polymerisation of enzymes leading to their inactivation and resulting in cellular damage. Symptoms occur above a threshold of 150 kPa of oxygen and include:

- Visual changes – particularly tunnel vision
- Tinnitus
- Nausea
- Twitching
- Irritability
- Dizziness
- Tonic-clonic seizures
- Unconsciousness.

There are several factors which predispose to CNS toxicity and they are:

- Exercise
- Cold
- Stress
- Fatigue
- Elevated PaCO_2
- Dietary deficiency of trace elements
- Nitrogen narcosis.

At rest in a dry environment patients can tolerate up to 280 kPa of oxygen given intermittently.

What are the ocular effects of oxygen toxicity and when do they occur most commonly?

Ocular or retinopathic conditions include:

- Reversible constriction of the peripheral field of vision
- Progressive reversible myopia
- Delayed cataract formation
- Retinopathy of prematurity.

Ocular effects are more common when the entire eye is exposed to high oxygen concentrations and hyperbaric pressures. For example, in an oxygen tent or hyperbaric chamber, rather than when there is hyperoxia of the arterial circulation.

What is retinopathy of prematurity?

Retinopathy of prematurity (ROP), originally named retrolental fibroplasia, is a condition of the eye seen in premature babies. Full maturation of the retinal blood vessels tends to occur close to 40 weeks gestation. When babies are born prematurely, retinal vessels are more prone to abnormal growth. Exposure to excess oxygen causes down-regulation of vascular endothelial growth factor (VEGF) leading to cessation of growth and an ischaemic peripheral retina. As the retina thickens it soon outgrows its blood supply, causing VEGF upregulation and proliferation of abnormal blood vessels. These vessels are prone to dilatation and rupture that subsequently leads to vitreal and retinal haemorrhages, fibrosis and adhesions, retinal detachment, and blindness.

Risk factors include:

- Degree of prematurity
- Low birth weight
- Duration of ventilation
- Duration of CPAP
- Severity of comorbidities at birth.

Supplemental oxygen exposure is a risk factor but restricting oxygen will not necessarily reduce the rate of progression and may have systemic implications. Concentrations of above 40% for 1–2 days after birth may be implicated and for neonates who develop ROP a PaO_2 of greater than 10.4 kPa for more than 6 hours may cause progressive damage, so an oxygen saturation of 90% is aimed for.

Are there any other toxic effects of oxygen you know about?

The main effects have been covered; this is small print and you are doing well if you are asked this!

Oxygen toxicity can also affect red blood cell morphology. A reduction in red cell mass can occur and haemolysis has been seen following hyperbaric oxygen therapy. Serous otitis media and dysbaric osteonecrosis have been observed in astronauts and are thought to be partially attributable to high oxygen concentrations while in space flights.

What levels of oxygen carry risk?

At atmospheric pressure, oxygen toxicity is related to the dose and time. However, there is a lot of individual variation in the development of toxicity. The safest policy is to wean patients off high oxygen concentrations as early as possible without compromising oxygen delivery.

Can you think of any specific circumstances where you might deliberately use high concentration oxygen therapy in the non-hypoxaemic patient?

High concentration oxygen therapy can be used in the following circumstances:

- CO poisoning
- Cyanide poisoning
- Pneumothorax

- Cluster headaches
- During the initial assessment / resuscitation of the critically unwell patient.

What are the indications for hyperbaric oxygen therapy?

Hyperbaric oxygen is oxygen administered at higher than atmospheric pressure. It may be considered as a therapeutic agent in the following circumstances, but judicious use is advised to balance for the recognised adverse effects of this therapy.

The following uses are recommended by the European Committee for Hyperbaric Medicine:

- Carbon monoxide poisoning
- Open fractures with crush injury
- Prevention and treatment of osteoradionecrosis
- Soft tissue radionecrosis
- Decompression sickness
- Gas embolism
- Anaerobic or mixed bacterial infection
- Sudden deafness
- Diabetic foot lesions
- Femoral head necrosis
- Central retinal artery occlusion.

The toxic effects are predominantly central nervous system effects. Under hyperbaric conditions, oxygen toxicity is again dose dependent and is the main limitation to treatment. It affects the CNS, lung, myocardium, liver, kidneys, and visual system. After 8 hours of receiving 100% oxygen at a pressure of 2 atmospheres, subjects may experience a decreased vital capacity, facial twitching, abnormal taste and smell, and tonic-clonic seizures. It may cause cardiovascular problems including vasoconstriction and myocardial depression.

Further Reading

- Brubakk A, Neuman T. *Bennett and Elliott's Physiology and Medicine of Diving*. Fifth Revised Edition. Edinburgh, New York: Saunders, 2003.
- BTS guideline for oxygen use in adults in healthcare and emergency settings. British Thoracic Society Emergency Oxygen Guideline Development Group. *Thorax*. 2017; 72 (1): 1–90.
- Davidson LM, Berkelhamer SK. Bronchopulmonary dysplasia: Chronic lung disease of infancy and long-term pulmonary outcomes. *Journal of Clinical Medicine*. 2017; 6 (1): 4.
- Floyd TF, Clark JM, Gelfand R, et al. Independent cerebral vasoconstrictive effects of hyperoxia and accompanying arterial hypocapnia at 1 ATA. *Journal of Applied Physiology*. 2003; 95(6): 2453–2461.
- Hofmann R, James SK, Jernberg T, et al. Oxygen therapy in suspected acute myocardial infarction. *New England Journal of Medicine*. 2017; 377: 1240–1249.
- Horncastle E, Lumb AB. Hyperoxia in anaesthesia and intensive care. *British Journal of Anaesthesia Education*. 2019; 19 (6): 176–182.
- Martin DS, Grocott MPW. Oxygen therapy and anaesthesia: Too much of a good thing? *Anaesthesia*. 2015; 70 (5): 522–527.
- Martin DS, Grocott MPW. Oxygen therapy in anaesthesia: The yin and yang of O₂. *British*

Journal of Anaesthesia. 2013; 111 (6): 867–871.

Mathieu D, Marroni A, Kot J. Tenth European consensus conference on hyperbaric medicine: Recommendations for accepted and non-accepted clinical indications and practice of hyperbaric oxygen treatment. *Diving and Hyperbaric Medicine*. 2017; 47 (1): 24–32.

Moradkhan R, Sinoway LI. Revisiting the role of oxygen therapy in cardiac patients. *Journal of the American College of Cardiology*. 2010; 56: 1013–1016.

Patel DN, Goel A, Agarwal SB, Garg P, Lakhani KK. Oxygen toxicity. *Journal of Indian Academy of Clinical Medicine*. 2003; 4(3): 234–237.

Roffe C, Nevalle T, Sim J et al. Effect of routine low-dose oxygen supplementation on death and disability in adults with acute stroke: The Stroke Oxygen Study randomised clinical trial. *Journal of the American Medical Association*. 2017; 318: 1125–1135.

Stub D, Smith K, Bernard S et al. Air versus oxygen in ST-segment-elevation myocardial infarction. *Circulation*. 2015; 131: 2143–2150.

3.2.6 Renal Replacement Therapies – Emily K Johnson and Antonia Stone

You are called to ITU to review a 46-year-old man. He is a known intravenous drug abuser and alcoholic who had been brought into the emergency department 24 hours previously. He had been found by a neighbour, unconscious and lying on the floor of his hallway. On admission his Glasgow Coma Score (GCS) score was 8 and his temperature 34°C. He was resuscitated. His blood results are as follows:

Sodium	141 mmol/L
Potassium	6.4 mmol/L
Urea	35.6 mmol/L
Creatinine	760 µg/L
Haemoglobin	85 g/L
White cell count	18 × 10 ⁹ /L
Platelets	150 × 10 ⁹ /L
Creatinine kinase (CK)	15600 IU

He has an ECG demonstrating a rate of 64 beats per minute, an increased PR interval and tall tented T waves.

This is a good example of a long clinical case you may be faced with. There are many potential routes of questioning and often numerous abnormal results with potential for discussion. You will have 10 minutes to study the case and make notes; use this time wisely, the information will be available at the exam table too so you do not need to copy it out. The examiners will lead you along their structured line of questioning but they will start by asking you for a summary. It is crucial that you come across well at this stage and a SUCCINCT summary is advisable. It is worth taking a few minutes to prepare your summary.

Can you summarise the case?

This is a 46-year-old man who is a known substance abuser and has been found collapsed on the floor for an unknown length of time. He has acute renal failure and life-threatening hyperkalaemia, which are most likely to be secondary to acute rhabdomyolysis.

It is better to give a short summary and let the examiner lead the questioning than go into the finer details of the case. It is likely a discussion about the investigations, particularly the causes of any abnormalities will follow.

What could be the causes of his acute kidney injury?

Acute kidney injury, previously known as acute renal failure, can be divided into pre-renal, intrinsic and post-renal causes. The most likely cause in this scenario is acute rhabdomyolysis. The patient has a history suggesting muscle damage may have occurred. This is associated with a significantly raised creatinine kinase, a biochemical marker of muscle breakdown.

Differential diagnoses may include the following:

Pre-renal causes: (caused by reduced blood flow to the kidney)

- Hypovolaemia due to haemorrhage, severe dehydration or burns
- Hypotension
 - Secondary to low cardiac output states, for example in cardiogenic shock, heart failure or massive PE
 - Secondary to systemic vasodilation due to sepsis, anaphylaxis or hepatorenal syndrome
- Renal vasoconstriction caused by NSAIDs or renovascular disease

Intrinsic renal causes: (those affecting the glomerulus or tubule)

- Acute tubular necrosis caused by drugs, rhabdomyolysis or ischaemia from prolonged pre-renal injury
- Acute interstitial nephritis caused by drugs or infection
- Autoimmune disease
- Toxins causing intratubular blockage such as ethylene glycol

Post-renal causes: (these are obstructive causes)

- Renal / ureteric calculi
- Prostate hypertrophy
- Tumour.

Do you know of any classifications for acute kidney injury?

If you don't know then just say so. Don't try to make up an answer to a question like this – it is unlikely that such a question alone is a pass or fail question.

While there is no universally accepted definition of AKI, research studies have proposed multiple classification systems; of which Kidney Disease: Improving Global Outcomes (KDIGO) is the most recent and frequently used.

1. RIFLE was the first widely accepted classification criteria for AKI. It is an acronym for risk, injury, failure, loss of kidney function, end-stage renal failure and uses serum creatinine or urine output to define severity of AKI. Parameters are as follows:
 - **Risk:** Serum creatinine raised 1.5 times baseline or urine output <0.5 ml/kg for 6 hours
 - **Injury:** Serum creatinine raised 2 times baseline or urine output <0.5 ml/kg for 12 hours
 - **Failure:** Serum creatinine raised 3 times baseline or urine output <0.3 ml/kg for 24 hours (or anuria for 12 hours)
 - **Loss:** persistent acute renal failure or complete loss of kidney function for more than 4 weeks
 - **End-stage renal disease:** complete loss of kidney function for more than 3 months
2. Acute Kidney Injury Network (AKIN) later published a modified version of RIFLE called AKIN criteria which instead divides the severity into stages 1, 2 or 3. It is based on the serum creatinine rise and urine output.
3. Most recently, KDIGO (Kidney Disease: Improving Global Outcomes) classified AKI severity according to the following criteria:
 - **Stage 1:** Serum creatinine 1.5–1.9 times baseline OR ≥ 0.3 mg/dL increase
Urine output <0.5 ml/kg/h for 6–12 hours
 - **Stage 2:** Serum creatinine 2.0–2.9 times baseline
Urine output <0.5 ml/kg/h for ≥ 12 hours
 - **Stage 3:** Serum creatinine 3.0 times baseline
OR increase in serum creatinine to ≥ 4.0 mg/dL
OR initiation of renal replacement therapy
OR in patient <18 years, decrease in eGFR to <35 ml/min
Urine output <0.3 ml/kg/h for ≥ 24 hours OR anuria for ≥ 12 hours.

It is worth considering that while serum creatinine is reciprocally related to GFR, levels only start to increase significantly once approximately 50% of the GFR has been lost. Hence, early recognition of renal dysfunction is extremely challenging.

Current research is investigating the role of novel biomarkers in the early detection of AKI which may impact future classification systems.

When this patient was first admitted, what are the treatment priorities?

On admission, my initial treatment priority would be immediate administration of oxygen and stabilisation of any airway, respiratory and circulatory abnormalities. Cervical spine stabilisation will also be required as he may have sustained trauma and would therefore need a full secondary survey. Treatment of his hyperkalaemia should be undertaken immediately as his potassium is dangerously high, as evidenced by the ECG changes. He should also be actively warmed as he is hypothermic.

The underlying cause of his unconsciousness should be sought and appropriately treated and while a secondary survey may help identify this, his history indicates blood toxicology may also be helpful.

How would you manage his hyperkalaemia?

I would initially manage his hyperkalaemia by giving 10 ml of 10% calcium gluconate intravenously with cardiac monitoring in situ; this will help to stabilise the myocardium by raising the threshold potential to excitation, thus reducing his susceptibility to ventricular arrhythmias. Next, I would commence an infusion of insulin (10 units of insulin with 50 ml of 50% dextrose given over 5 minutes) to help shift K^+ back into cells. Salbutamol 10–20 mg in 4 ml saline via a nebuliser would also have this effect. In addition, I could consider a cation exchange resin such as a 30 mg enema of calcium resonium, although their effectiveness remains debatable. I would recheck his serum potassium and if it is not responding to conservative treatment, I would consider definitive treatment with haemofiltration.

What are the indications for starting haemofiltration?

The main indications for starting haemofiltration can be defined as renal or non-renal:

- **Renal**

- Refractory hyperkalaemia (>6.5 mmol/L or rapidly rising)
- Refractory metabolic acidosis ($pH < 7.15$)
- Fluid overload resistant to diuretic therapy
- Symptomatic uraemia (encephalopathy, pericarditis, blood dyscrasias)

- **Non-renal :**

- Toxin ingestion (if toxin is cleared by dialysis)
- Hyperthermia or rewarming of a hypothermic patient
- Hyponatraemia >160 mmol/L

There are no universally agreed thresholds at which haemofiltration should be commenced and there is much debate around the timing for renal replacement therapy. Some studies suggest early initiation of RRT may correlate with reduced mortality in critically ill patients.

Haemofiltration may also be employed for the treatment of dysnatraemias and for plasmapheresis. Emerging evidence suggests haemofiltration may be indicated in the management of systemic inflammatory response syndrome (SIRS) to reduce the plasma levels of inflammatory mediators.

Can you give examples of drugs cleared by dialysis?

Drugs that have a low volume of distribution, low molecular weight and low plasma protein binding are more likely to be removed from plasma by dialysis.

Examples include:

- Ethylene glycol
- Methanol
- Salicylate (aspirin)
- Vancomycin
- Theophylline
- Lithium

Can you classify the types of renal replacement therapies?

Renal replacement therapies can be classified into intermittent and continuous techniques. Intermittent dialysis can be either intermittent haemodialysis or peritoneal dialysis. Continuous renal replacement therapies are more commonly used in the critical care setting. They consist of continuous veno-venous haemofiltration (CVVH), continuous veno-venous haemodialysis (CVVHD), continuous veno-venous haemodiafiltration (CVVHDF) and slow continuous ultrafiltration (SCUF).

Can you tell me a bit more about the intermittent techniques of renal replacement therapy?

This is unlikely to be asked but it is helpful for your understanding of the subject.

Intermittent renal replacement therapies are most commonly used in chronic renal failure where the patients are haemodynamically stable.

Intermittent haemodialysis requires insertion of specialist dialysis catheters or the formation of an arterio-venous fistula. Dialysis takes place at regular intervals, depending on the degree of renal impairment, commonly three times a week. It involves large volumes of blood leaving the circulation at any one time so rapidly removes a solute or volume load, but is unsuitable if a patient is already haemodynamically compromised. The primary method of solute and fluid removal in haemodialysis is diffusion.

Peritoneal dialysis uses the peritoneum as the dialysis membrane and fluid can be introduced at regular intervals throughout the day, which is continuous ambulatory peritoneal dialysis (CAPD) or overnight using automatic peritoneal dialysis (APD). This form of dialysis requires a permanent peritoneal catheter, which carries a considerable risk of infection. It is also inefficient at removing large volumes of fluid or solute and would be inappropriate in critically ill patients as the increase in intra-abdominal volume could cause splinting of the diaphragm and ventilation difficulties.

What are the differences between the types of continuous renal replacement therapies?

The continuous renal replacement therapies are based on the transfer of solute and fluid across a semipermeable membrane. This occurs either by diffusion in haemodialysis or by convection in haemofiltration.

In haemodialysis, the dialysate fluid is pumped in the opposite direction to blood, allowing rapid clearance of smaller waste molecules as they diffuse across the semipermeable membrane via their waste-solute concentration gradient.

In haemofiltration, plasma, water and solute pass through the semipermeable membrane driven by a hydrostatic pressure gradient in a process called convection. It occurs more slowly than haemodialysis as there is no diffusion taking place.

Haemodiafiltration is a combination of these two processes.

What is the difference between diffusion, osmosis and dialysis?

Diffusion is the spontaneous net movement of a substance across a semipermeable membrane from a region where it is in a high concentration to a region where it is in a low concentration. The rate of diffusion is proportional to the concentration gradient.

Osmosis is the diffusion of a liquid solvent (water) from a weak solution across a semipermeable membrane into a stronger solution so that when equilibrium is reached the osmotic pressures on each side are equal.

Dialysis is the separation of substances in solution by means of their unequal diffusion through semipermeable membranes.

What determines the rate of haemodialysis?

In haemodialysis, the rate of diffusion is driven by the difference in solute concentration between the blood and dialysate: the larger the concentration gradient, the **faster** the rate of diffusion.

Other factors influencing the rate of diffusion relate to the *solute* itself, including particle size, ionic charge and degree of plasma protein binding. The properties of the *dialysis membrane* also influence diffusion rate including type, thickness, surface area and number of pores.

Dialysis becomes more efficient as the rate of delivery and concentration of dialysate increases.

What is convection?

The mechanism of convection reflects the process that occurs within the renal corpuscle, it is also known as 'solvent drag'.

Appropriately sized solute molecules are swept through the semipermeable membrane by a moving stream of ultrafiltrate, driven by a pressure difference between the compartments.

Unlike diffusion, it is independent of solute concentration and instead, the direction and force of the transmembrane pressure (TMP) dictates solute clearance.

What factors influence the rate of convection?

The rate of convective transport is determined by the direction and magnitude of the transmembrane pressure. The porosity of the membrane also influences the rate and degree at which solutes pass through.

The hydrostatic pressure in the blood compartment is dependent on the rate of blood flow, therefore increasing blood flow will increase the rate of ultrafiltration and solute clearance.

Similarly, increasing the negative pressure in the ultrafiltrate compartment, will also increase the rate of ultrafiltration.

This means that increased hydrostatic pressure in the blood will increase transmembrane pressure (TMP) and speed up ultrafiltration; similar effects would occur with a reduction in the plasma colloid osmotic pressure.

Could you draw a simple circuit for continuous veno-venous haemofiltration (CVVH)?

This could well be expected, and it is recommended you spend a bit of time practising a simple diagram of a haemofiltration circuit (Figure 3.2.6). You need to understand the differences between CVVH and CVVHDF circuits and be able to illustrate these on your diagram. CVVH relies on the process of convection, and CVVHDF relies on both convection and diffusion.

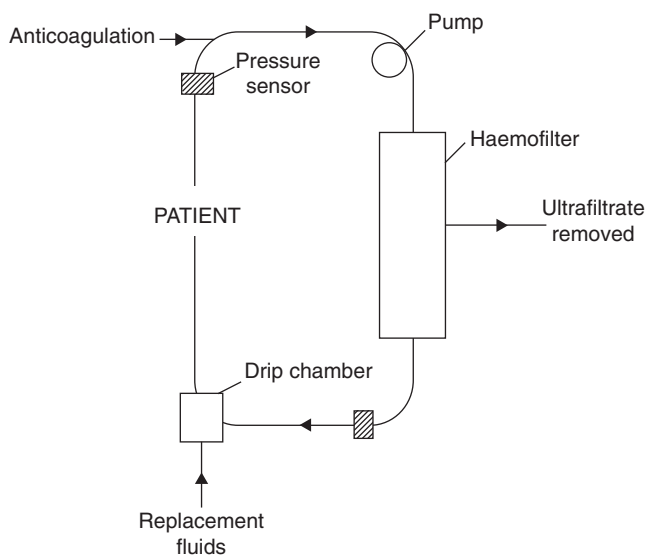


Figure 3.2.6 Schematic diagram of a haemofiltration circuit.

A CVVH or haemofiltration circuit starts with central, large bore intravenous access to the patient. A single cannula has a dual lumen for removal and return of blood. The removal flow may range between 0 and 300 ml/min. This will pass through a pressure sensor to detect changes in pressure that may indicate cannula blockage. Then a volumetric pump drives the flow of blood through the haemofilter where the ultrafiltrate is removed and delivered into a collection bag. The filtered blood continues in the circuit through another pressure sensor. The blood then passes into a drip chamber where replacement fluids can be added. Blood is then returned to the patient via a final pressure sensor.

The rate of ultrafiltration, and therefore of the solute convection, depends on the speed of the pump and the resultant transmembrane pressure generated.

In summary the components of a CVVH circuit are:

- Double-lumen central venous catheter
- Afferent and efferent tubing
- Pressure sensors
- Volumetric pump
- Haemofilter and drip chamber
- Collection bag and tubing for ultrafiltrate
- Portal for anticoagulants and replacement fluids.

Describe how this differs from continuous veno-venous haemodiafiltration (CVVHDF)?

In CVVHDF there is dialysate entering the haemofilter. This is because dialysis is used in addition to haemofiltration. Dialysate fluid is pumped through to maintain concentration gradients on either side of the semipermeable membrane and therefore allow solute transfer by diffusion. These two solutions must run in opposite directions to

allow convection to occur as well. The pump blood flow is much higher, ranging from 200 to 400 ml/min making this a more efficient method of continuous renal replacement therapy.

Can you tell me what types of membranes are used in continuous renal replacement therapy?

There are two types of membrane used in renal replacement therapy, natural (cellulose) and synthetic (polyacrylonitrile). The interaction between the blood and membrane is known as biocompatibility; the more biocompatible the membrane is, the less likely it is to cause harmful side effects. Most modern haemofilters use synthetic membranes as they are considered more biocompatible, allow the clearance of larger molecules and are associated with better patient outcomes. Cellulose-based membranes can trigger inflammation and are considered less biocompatible.

What are the different types of replacement fluids commonly used?

During haemofiltration, bicarbonate ions will need to be replaced because they are freely filtered out. The replacement fluids consist of balanced solutions containing phosphate and potassium with either lactate or bicarbonate as a buffer.

Lactate containing solutions were previously used as standard as they are cheaper and have a longer shelf life. However, as lactate is hepatically metabolised, it can cause hyperlactataemia and worsen a metabolic acidosis, particularly in patients with liver dysfunction or a pre-existing raised lactate.

An alternative is a bicarbonate-based solution; evidence suggests it may confer improved control of acidosis and cardiovascular stability. This can be added to the circuit before the haemofilter (pre-dilution), which reduces the chance of the filter clotting but is less efficient, or mixed with blood in the drop chamber (post-dilution), which is more efficient at clearing solutes but also more likely to reduce the lifespan of the filter by clotting off.

What are the options for anticoagulation when haemofiltering a patient on ICU?

Anticoagulation can be either systemic or regional and is required in haemofiltration due to the risk of clot formation.

Systemic heparin anticoagulation is used most commonly as it is cheap, readily available and it is easy to test its activity on a coagulation screen. It is used initially to flush the filter, then as a bolus to start the filter followed by an infusion while the filter is running. As it is systemic anticoagulation, patients are at risk of bleeding. There is also the risk of heparin-induced thrombocytopenia (HIT) and heparin resistance.

Citrate is a popular alternative as it is confined to the haemofilter circuit, thus avoiding systemic anticoagulation. It acts by chelating calcium ions, inhibiting platelet aggregation and coagulation. The calcium is then replaced after filtering to reverse this effect and residual citrate is rapidly metabolised by the liver. It does not cause HIT and evidence suggests it increases filter lifespan compared with heparin. However, it can cause metabolic and electrolyte disturbances and it does not provide venous thromboembolism prophylaxis.

An alternative option is the prostacyclin infusion Flolan™ which inhibits platelet aggregation and can be used in patients with pre-existing thrombocytopaenia. Low molecular weight heparin, fondaparinux and danaparoid sodium may also be used and patients with a pre-existing coagulopathy may not require anticoagulation at all.

What are the potential complications of CVVH?

The complications of CVVH can be divided into those associated with the intravenous catheter itself, those associated with anticoagulation and those associated with the filtration process.

- **Catheter-related:**

Complications at insertion (these are site dependent and the same as those for a standard central line, bearing in mind the intravenous catheters for haemofiltration are significantly larger bore than a standard central line).

- Bleeding
- Infection
- Damage to surrounding structures
- A-V fistulae
- Arrhythmias
- Pneumothorax
- Pain

- **Anticoagulation-related:**

- Bleeding
- Heparin-induced thrombocytopaenia

- **General complications:**

- Cardiovascular instability and hypotension, particularly when starting filtration on a critically ill patient
- Hypovolaemia
- Hypothermia
- Electrolyte imbalance
- Metabolic abnormalities
- Air embolism
- Anaemia
- Reactions to the filter membrane such as anaphylaxis.

Can you tell me more about the role of renal replacement therapies in sepsis?

It has been proposed that septic patients may benefit from removal of pathogenic, inflammatory mediators by high volume haemofiltration (HVHF).

The most recent evidence suggests that high volume haemofiltration (effluents greater than 50 ml/kg/hr) can lead to a reduction in vasopressor requirements but does not influence mortality or long-term dialysis dependency. No study has yet demonstrated a consistent reduction in serum cytokine concentration with HVHF and there

may be an inadvertent risk of reduced plasma concentration of antibiotics. The cost effectiveness of this method also remains in question.

Further Reading

- Bellomo R, Kellum JA, Ronco C. Defining acute renal failure: Physiological principles. *Intensive Care Medicine*. 2004; 30: 33–37.
- Borthwick E, Hill C, Rabindranath K, Maxwell A, McAuley D, Blackwood B. High-volume haemofiltration for sepsis in adults. *Cochrane Database of Systemic Reviews*. 2017; 31(1).
- Gemmell L, Docking R, Black E. Renal replacement therapy in critical care. *British Journal of Anaesthesia Education*; 2017; 17 (3): 88–93.
- Goren O, Matot I. Perioperative acute kidney injury. *British Journal of Anaesthesia*. 2015; 115(2): 3–14.
- Hall N, Fox A. Renal replacement therapies in critical care. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2006; 6(5): 197–202.
- Nyirenda M, Tang J, Padfield P, Seckl J. Hyperkalaemia clinical review. *British Medical Journal*. 2009; 339: 1020–1024.
- Ricci Z, Cruz D, Ronco C. The RIFLE criteria and mortality in acute kidney injury: A systematic review. *Kidney International*. 2008; 73: 538–546.

3.2.7 Organ Donation – Philip Harrington

The diagnosis and evaluation of brainstem death is covered elsewhere in this book and so will not be covered here.

You are looking after a 60-year-old patient on the intensive care unit who has suffered a prolonged cardiac arrest in the community with a return of spontaneous circulation after advanced life support was delivered in the emergency department. They have been mechanically ventilated on ICU for 5 days and despite receiving no sedation for the last 2 days they have not woken but will take occasional spontaneous breaths. All appropriate investigations have been completed and the outcome is very guarded.

You speak to the family and explain the likelihood of a poor outcome; they understand, have been expecting this and ask you about the possibility of organ donation for their relative.

Under what circumstances may organ donation occur?

Organ donation may occur after following one of two general pathways resulting in legal death with the patient being declared dead by either cardiorespiratory or neurological criteria. These are termed donation after circulatory death (DCD) and donation after brain death (DBD). Both pathways will involve and be overseen by a specialist nurse in organ donation (SNOD) and the processes surrounding each pathway are different.

For DBD a patient will undergo brainstem death testing and be declared legally dead. They will then undergo a period of medical optimisation prior to a retrieval operation where organs will be procured by a surgical team and prepared for transport and implantation into a donor.

For DCD death is confirmed using classical cardiorespiratory criteria. Donation may be controlled (where death is expected and life sustaining treatment is withdrawn) or uncontrolled (following unexpected cardiac arrest). The Maastricht criteria explain the potential circumstances (see Table 3.2.7).

Table 3.2.7 Maastricht classification for donation after cardiac death

Category I	Dead on arrival to hospital	Uncontrolled
Category II	Unsuccessful resuscitation	Uncontrolled
Category III	Awaiting cardiac arrest	Controlled
Category IV	Cardiac arrest in a brainstem-dead donor	Controlled
Category V	Unexpected cardiac arrest in a critically ill patient	Uncontrolled

Typically in UK clinical practice Category 3 will be encountered; in an appropriate situation planned withdrawal of life-sustaining treatment occurs, death is confirmed by cardiorespiratory criteria and the patient transferred to theatre for a retrieval operation.

Are there any contraindications to organ donation?

There are absolute and relative contraindications to organ donation. Absolute contraindications include variant CJD infection and active HIV disease (differing from controlled HIV infection).

Relative contraindications include conditions that will likely render a patient unsuitable as a donor as per NHSBT (NHS blood and transplant). These include, but are not limited to, active cancer in the last 3 years, primary intracerebral lymphoma, secondary intracerebral tumours, haematological malignancy, untreated systemic infection, active and untreated tuberculosis.

When considering Donation after Cardiac Death:

What is warm ischaemic time?

Warm ischaemic time is the time from asystole to the onset of cold organ perfusion established once the organ has been retrieved; it is important as cold perfusion aims to reduce the metabolic demand of the organ. Different organs have different warm ischaemic tolerance before they are unable to be transplanted once donated due to irreparable damage from ischaemia. However, once withdrawal of life sustaining treatment occurs death is rarely rapid and so functional warm ischaemic time is referred to, defined by the time from systolic blood pressure of less than 50 mmHg to cold organ perfusion.

Can you outline the process of donation?

The first step in the process is early referral to the local transplant coordinator who will be able to advise as to suitability and also check the patient's registration on the organ donor register. As of May 2020 the process for this in the UK changed to an opt-out system whereby adults in England are considered to have agreed to be an organ donor at death unless they have specifically requested to not donate. Families are still able to decline organ donation at any stage of the process. The transplant coordinator or SNOD will then attend the unit and help to facilitate discussions with the family about the ongoing process of donation. Ongoing medical care should be considered with the same

level of critical care support being continued but any escalating measures that could be considered to cause the patient harm or distress should not be implemented.

Once the surgical team is on-site and ready in the receiving theatre then the process of withdrawal of treatment should be commenced in line with the unit's usual policy. It should be noted during this stage that the process may have to be abandoned if the dying process is prolonged due to excessive warm ischaemic time. This time will differ based on which organs are being donated.

Once cardiac arrest has occurred then a medical practitioner separate from the retrieval team should confirm death based on cardiorespiratory criteria and should examine the patient for the usual 5 minute duration. Once death has been confirmed then relatives may spend up to 5 minutes with the patient; following this the patient will be transferred to the operating theatre to undergo the retrieval operation. Regardless of whether the family opt to spend time with their loved one the retrieval operation does not start until 10 minutes since the loss of a circulation.

What perioperative considerations are there?

Lung donation may require re-intubation to prevent aspiration and reduce hypoxia. However, conventional ventilation risks resuscitation and re-establishing a circulation. Practically, once re-intubated a single recruitment manoeuvre is performed followed by the application of CPAP via the ventilator. Importantly this should not happen until 10 minutes has elapsed since mechanical asystole. Ventilation should not be restarted until the cerebral circulation has been surgically isolated.

DCD heart donation in the UK occurs via re-establishing coronary perfusion once the organ is either isolated within the donor or removed from the donor depending on the technique being used. The heart will then be transported perfused to the recipient. This will all be coordinated and performed by the retrieval team.

When considering Donation after Brain Death:

How can medical management of DBD patients be optimised?

Brainstem death induces a number of physiological insults and active management of changes that occur is key to maximising the number of transplantable organs from the donor. There are general and organ specific principals to consider.

From a general care perspective the patient should be managed on the intensive care unit ; not only does this allow specialist nursing and medical care but also allows support for relatives during the process. Minimum invasive monitoring includes arterial and central venous pressure monitoring with consideration of cardiac output monitoring. Unnecessary drugs should be stopped and normothermia should be maintained.

From a respiratory standpoint lung-protective ventilation should be provided with tidal volumes of 6–8 ml/kg and optimal PEEP to minimise oxygen requirement. Recruitment manoeuvres may be used as required and fluid overload should be avoided. This can be done by minimising intravenous fluids and using diuretics if needed.

Cardiovascular parameters can be adjusted in accordance with cardiac output monitoring and specific targets that may be set by the retrieval team. Vasopressin is the drug of choice to maintain vascular tone with the aim of reducing exogenous catecholamine use.

Intravenous fluids may be given as required but a positive fluid balance should be avoided. Urine output is targeted in the 0.5 to 2.5 ml/kg range – if over 4 ml/kg then the

presence of diabetes insipidus should be considered, investigated and treated. The patient's feeding regime should be continued and blood glucose kept within the normal range. Electrolytes derangement should be corrected as necessary.

From an endocrine perspective methylprednisolone is given to aim for haemodynamic stability and improve respiratory function while triiodothyronine (synthetic T3) may be considered as directed by the retrieval team. Standard thromboprophylaxis should be maintained.

The retrieval team may have specific investigation requests for the donor which may include an up-to-date ECG, echocardiogram, coronary angiogram, broncho-alveolar lavage and chest X-ray.

What is cold ischaemic time?

Cold ischaemic time is defined as the period from the start of cold perfusion of an organ after cessation of circulation to the time of the start of the first vascular anastomosis at implantation.

What perioperative factors need to be considered?

The retrieval operation may involve a midline laparotomy plus sternotomy depending on which organs are to be donated. Issues may include significant blood loss, heat loss and haemodynamic lability and maintaining stability is key to a successful retrieval of organs in optimal condition.

In addition spinal reflexes are common; importantly they do not mean a return of brain function and may require neuromuscular blockade for optimal surgical conditions.

Further Reading

Corbett S, Trainor D. Perioperative management of the organ donor after diagnosis of death using neurological criteria *British Journal of Anaesthesia* 2020; 21(5): 194–200.

Dunne K, Doherty P. Donation after circulatory death *British Journal of Anaesthesia*. 2011; 11(3): 82–86.

Flood S, Torgoff C. A new heart for organ donation after circulatory death *British Journal of Anaesthesia*. 2020; 20(4): 126–132.

3.2.8 COVID-19 – Philip Harrington

This topic may be asked as a short clinical case or as part of a wider discussion about a long case.

What is COVID-19?

COVID-19 is an infectious disease that is caused by the virus named SARS-CoV-2. This is a coronavirus that is genetically related to the viruses that caused the SARS and MERS outbreaks. It is considered to be zoonotic in origin and caused a global pandemic in 2020.

COVID-19 primarily spreads by droplet and aerosol means and can be passed on via actions such as breathing, speaking, coughing and sneezing. In healthcare environments aerosol generating procedures such as certain kinds of investigations and assessments, non-invasive respiratory support as well as airway management can confer a higher risk of virus spread.

How does COVID-19 present?

Different variants of COVID-19 formed by various small mutations can alter the virus' incubation period. Generally, however, this is in the range of 3–8 days. Importantly up to a third of people infected with the virus may show no symptoms.

Typical symptoms include fever, cough and loss of taste and smell. Other systemic effects may involve shortness of breath and respiratory compromise, general malaise, loss of appetite, myalgia and any other symptoms usually associated with a general viral illness.

Older patients and the immune compromised may present in a non-typical fashion with reduced mobility, confusion and delirium amongst others. Children may be asymptomatic or experience milder symptoms than the adult population. In rare circumstances children may be affected by the severe paediatric multisystem inflammatory syndrome temporally associated with SARS-CoV-2 (PIMS-TS). This may begin as a regular viral illness but subsequently manifest as a severe multisystem inflammatory condition that can involve multiple organs and mimic other disease states such as Kawasaki Disease, sepsis and toxic shock syndrome.

What complications may occur?

The majority of patients who are symptomatic developed only mild or moderate disease and this is particularly true for the vaccinated population.

A small percentage of patients, however, may suffer severe disease with acute respiratory failure, acute respiratory distress syndrome (ARDS), shock, multiorgan failure and the development of venous thromboembolism. Other organ manifestations may include acute kidney injury and cardiac involvement in the form of myositis.

The general complications of severe multiorgan failure, mechanical ventilation and prolonged intensive care admission must also be considered.

Who is at risk of COVID-19?

Risk factors for patients suffering severe disease include those with advanced age, men, the obese, patients from certain ethnic backgrounds such as the black and Asian communities and also patients from socio-economically deprived areas. Those that have baseline significant comorbidities are also at risk as well as those suffering from a more severe variant strain of the virus.

You are asked to see a COVID-19 positive patient who needs a laparotomy on the emergency list.

What infection control considerations may be required for this patient?

COVID-19 infection in hospitalised patients should be identified via nasal and/or oropharyngeal/tracheal sample. Emergency admissions should be tested on arrival regardless of symptoms and those who develop symptoms should be retested. Once identified as positive for COVID-19 the patient should be cared for in an appropriately cohorted area to reduce the risk of spread to patients who are negative.

Personal protective equipment should be used in these areas which will consist of a surgical face mask and apron at a minimum. If aerosol generating procedures are to be performed, such as in the theatre environment then an FFP3 mask, disposable gown and

gloves are recommended. If ongoing aerosol-generating treatments such as the use of non-invasive ventilation are required then the use of a negative pressure side room may be needed if cohorting patients is not possible.

Current recommendations suggest that the anaesthetic room may be used for induction with appropriate signage on the doors to prevent entry without the correct PPE. An available runner may be utilised to collect extra equipment required as it is recommended that the staff do not move in and out of the theatre during the case. If it is appropriate to wake the patient, they will need to be extubated and recovered in theatre until ready to go back to an appropriate cohorted area for ongoing care. If they are to remain sedated and ventilated then the intensive care unit will need to be liaised with to ensure the correct swab has been taken and that they can provide an appropriate side room or a bed in an appropriate COVID positive area prior to transfer.

Hand hygiene is paramount in the time surrounding the case and the theatre will need to undergo an extended cleaning procedure before being able to receive a subsequent patient.

Postoperatively the patient is extubated successfully and begins to recover from their abdominal surgery. Unfortunately 2 days later they deteriorate from a respiratory point of view and a diagnosis of COVID pneumonitis is considered.

What is the management of severe COVID-19?

Many patients will require no treatment and will recover at home needing over the counter remedies at most.

First of all severe COVID-19 infection needs to be diagnosed. Features that may suggest severe infection include clinical examination, oxygen requirement and work of breathing, chest X-ray and CT imaging indicating bilateral patchy infiltration and blood tests revealing low lymphocyte count, raised troponin, d-dimer, ferritin and C-reactive protein (CRP). In this patient other causes of respiratory deterioration should be considered including postoperative atelectasis or pneumonia, respiratory compromise due to pain at the operative site and pulmonary embolism. These should be investigated and ruled out as their treatment may diverge from the treatment of COVID-19.

Specific treatment for the respiratory complications of COVID-19 starts with the delivery of oxygen titrated to effect and clinical improvement. In patients requiring oxygen then pharmacological therapy with dexamethasone 6 mg once daily has been shown to reduce deaths. Similarly treatment with interleukin-6 receptor antagonists have been shown to reduce the risk of death in hypoxic and hospitalised patients who have a CRP above 75 mg/l; the available agents include sarilumab and tocilizumab.

High flow nasal oxygen and non-invasive ventilation may be considered based on the examination and clinical condition of the patient while considering that intubation and mechanical ventilation may be required. If the patient is compliant and appropriately ambulant then awake prone positioning is recommended to improve ventilation-perfusion matching and respiratory function.

If these measures fail then invasive mechanical ventilation may be required. It is important to adhere to lung-protective measures when invasively ventilating these patients and different trusts may have different protocols on various modes of mechanical ventilation including a regular bi-level ventilation strategy and inverse ratio ventilation. No single mode has been shown to improve outcomes, however. In the face of

worsening oxygenation ($\text{PaO}_2\text{:FiO}_2$ ratio <20 kPa with $\text{fiO}_2 >0.6$) then an early infusion of neuromuscular blocking agent as well as prone positioning are recommended. The prone position should be maintained for 16 continuous hours in order to have a mortality benefit. Further deterioration in appropriate patients should prompt the early referral to a centre offering veno-venous ECMO for extra corporeal respiratory support.

Further Reading

Faculty of Intensive Care Medicine, Intensive Care Society, Association of Anaesthetists, Royal College of Anaesthetists 2020. Clinical guidance. <https://icmanaesthesiacovid-19.org/clinical-guidance>.

Faculty of Intensive Care Medicine, Intensive Care Society, Association of Anaesthetists, Royal College of Anaesthetists 2020. Clinical guide for the management of critical care for adults with COVID-19 during the Coronavirus pandemic. [https://icmanaesthesiacovid-19.org/clinical-guide-for-the-management-of-critical-care-for-](https://icmanaesthesiacovid-19.org/clinical-guide-for-the-management-of-critical-care-for-adults-with-covid-19-during-the-coronavirus-pandemic)

[adults-with-covid-19-during-the-coronavirus-pandemic](https://icmanaesthesiacovid-19.org/clinical-guide-for-the-management-of-critical-care-for-adults-with-covid-19-during-the-coronavirus-pandemic).

National Institute for Health and Clinical Excellence (NICE). COVID-19 rapid guideline: Managing COVID-19. 2022. www.nice.org.uk/guidance/ng191/resources/covid19-rapid-guideline-managing-covid19-pdf-51035553326.

Perkins G, Ji C, Connolly B. Effect of noninvasive respiratory strategies on intubation or mortality among patients with acute hypoxemic respiratory failure and COVID-19 *Journal of the American Medical Association*. 2022; 327(6): 546–558.

Anatomy

4.1.1 Anatomy of Central Venous Access – Catherine Challifour

What are the indications for central venous access?

These can be diagnostic or therapeutic.

Diagnostic indications include:

- Measurement of central venous pressure
- Cardiac output monitoring and flotation of pulmonary artery catheters
- Measurement of mixed venous saturations.

Therapeutic indications include:

- Administration of drugs that are irritant if given peripherally (this includes some vasopressors and inotropes, total parenteral nutrition and many chemotherapy agents)
- Facilitate administration of multiple drugs by bolus or infusion
- Facilitate renal replacement therapy
- Facilitate plasma exchange therapy
- Transvenous pacing
- To enable intravenous therapies for patients with poor peripheral access or patients requiring long-term IV access.

Which sites are commonly used for central venous access?

The most commonly used sites for central venous catheters (CVC) are the internal jugular, subclavian and femoral veins. Peripherally inserted central catheters (PICC) use the cephalic, basilic or brachial veins accessed in the antecubital fossa to cannulate the superior vena cava.

What are the advantages and disadvantages of the different sites used?

The internal jugular vein is relatively easy to access and has a lower risk of pneumothorax compared with the subclavian route and a lower risk of infection when compared with the femoral vein.

The subclavian vein is more difficult to access due to its proximity to the clavicle and the procedure has a relatively higher rate of iatrogenic pneumothorax due to the proximity to the apex of the lung. Should inadvertent arterial puncture occur, compression of the subclavian artery can be challenging. However, this site has the lowest rate of infection and is well tolerated by patients needing longer-term access.

The femoral vein can be used when the internal jugular is not accessible (for example in head and neck surgery) or in patients with raised intracranial pressure who should not be positioned head down. It can be used when other routes of central access have been exhausted and there is no risk of pneumothorax. It does, however, have the highest rate of infection due to the proximity to the perineum.

Can you describe the anatomy of the internal jugular vein?

Anatomy questions are best answered using a simple diagram and a systematic approach to describe the relationships of the structure you are describing to local bones, muscles, ligaments, nerves and vasculature.

The internal jugular vein is a continuation of the jugular bulb; it extends from the jugular foramen and joins with the subclavian vein to form the brachiocephalic vein posterior to the sternoclavicular joint. The internal jugular vein lies within the carotid sheath along with the carotid artery and the vagus nerve. The relation of the internal jugular vein relative to the carotid artery changes throughout its course in the neck – initially it lies posterior to the artery before moving laterally and then finally anterolaterally.

The following structures lie superficially to the carotid sheath:

- Sternocleidomastoid muscle
- Platysma muscle
- Subcutaneous tissue
- Skin.

The following structures lie deep to the carotid sheath:

- Prevertebral fascia
- Vertebral muscles (scalene muscles and longus colli)
- Sympathetic chain
- Transverse processes of the cervical vertebrae.

At the root of the neck the dome of the pleura lies close to the internal jugular vein and on the left side, the thoracic duct lies posteriorly.

The ninth (glossopharyngeal), tenth (vagus) eleventh (accessory) and twelfth (hypoglossal) cranial nerves, the common and internal carotid arteries, the trachea and oesophagus lie medial to the internal jugular vein throughout its caudal course.

Can you describe the anatomy of the subclavian vein?

The brachial and basilic veins merge to form the axillary vein which passes medially from the axilla alongside the axillary vein and the brachial plexus within a fascial sheath. It runs posterior to the clavicle and becomes the subclavian vein at the lateral border of the first rib. It crosses the first rib in the subclavian vein groove and sits upon the pleura where it merges with the internal jugular vein to become the brachiocephalic vein.

The following structures lie posterior to the subclavian vein:

- Phrenic nerve
- Vagus nerve
- Anterior scalene muscle
- Subclavian artery.

Different techniques have been described for securing subclavian venous access but the subclavian vein is most easily accessed inferior to the clavicle between the midclavicular level and the junction of the medial and middle thirds of the clavicle.

Can you describe the anatomy of the femoral vein?

The femoral vein is a continuation of the popliteal vein and drains blood from the lower limb. The right and left femoral veins combine to form the inferior vena cava.

To gain central venous access the femoral vein is approached within the femoral triangle. The femoral triangle is a triangular fascial space within the proximal anterior thigh. The borders of the femoral triangle are the inguinal ligament superiorly, the sartorius muscle laterally and the medial border of adductor longus medially. The roof of the femoral triangle is formed by the fascia lata and the floor of the triangle is iliopsoas, pectineus and adductor longus muscles.

The femoral triangle contains the lateral cutaneous nerve of the thigh, the femoral branch of the genitofemoral nerve, the femoral nerve, the femoral artery and the femoral vein. In the superior aspect of the femoral triangle the femoral vein lies medially to the femoral artery, inferiorly the vein and artery may overlie each other and attempting to access the vein at this level may increase the risk of arterial puncture.

What is the role of ultrasound in securing central venous access?

Central venous access can be secured using either a surface anatomical landmark technique or under ultrasound guidance.

The use of real-time 2-D ultrasound allows an assessment of the vein diameter, patency and relation to surrounding structures. Non-pulsatile, compressible veins can be easily distinguished from pulsatile non-compressible arteries and thus the risk of inadvertent arterial puncture (or cannulation) can be minimised. Ultrasound enables visualisation of the needle entering the vein and can be used to confirm the correct placement of both the guidewire and the catheter within the vessel.

NICE guidance now recommends that the use of two-dimensional ultrasound guidance should be considered in most clinical circumstances where CVC insertion is necessary either electively or in an emergency situation. There is good evidence that the technique allows for successful cannulation with fewer attempts and reduces the risks of complications when compared with the landmark technique.

How can correct placement of a central venous catheter be confirmed?

On placement of the CVC, blood should be aspirated from each lumen of the catheter to ensure all lumens lie within the vessel.

Successful cannulation of the vein should be confirmed by the use of paired blood gases from both an arterial line and the newly sited central venous catheter – a significantly lower oxygen saturation value in the sample from the CVC confirms venous placement. The CVC should also be transduced to ensure a venous pressure waveform is seen.

For central venous catheters placed in the internal jugular or subclavian veins a chest X-ray should be performed to assess positioning of the catheter and to exclude iatrogenic pneumothorax during the catheter placement. The tip of a CVC placed in the internal jugular or subclavian vein should lie in the mid-section of the superior vena cava

(SVC) – more distal placement increases the risk of arrhythmias and cardiac tamponade should right atrial perforation occur. For catheters insufficiently advanced into the vein there is a risk of extravasation of drugs and fluids into the soft tissues from the most proximal lumens of the catheter. On chest X-ray the level of the carina corresponds with the mid-section of the SVC and the line tip should be seen at or just above the level of the carina.

What are the complications of central venous access?

Complications can occur at the time of insertion or may be delayed.

Immediate complications associated with line insertion include:

- Pneumothorax
- Air embolus
- Haemorrhage
- Haematoma formation
- Arterial puncture (and possible catheterisation)
- Arrhythmia
- Chylothorax (left-sided CVC only)
- Foreign body embolisation (guide wire or catheter fragments).

Delayed complications associated with the presence of a central venous catheter:

- Localised infection
- Line sepsis and bacteraemia
- Thrombosis
- Embolisation of thrombus
- Vessel stenosis
- Vessel erosion and perforation.

How can the risk of CVC-associated infection be minimised?

Central venous catheter-associated sepsis is the most common serious complication of catheter placement and it is vital that precautions are taken at the time of insertion and during ongoing use of the catheter to minimise this. Line site selection and avoidance of the femoral site where possible is important and the role of ultrasound to minimise the attempts required to place the catheter also plays a role. Antimicrobial-impregnated catheters are now in common use and should be selected when the catheter is anticipated being used for more than 5 days.

Insertion of a CVC should be undertaken as a sterile procedure. The operator should undertake effective hand washing in 4% chlorhexidine surgical scrub solution prior to donning a hat, mask, surgical gown and gloves. The patient's skin should be cleaned with aqueous chlorhexidine and alcohol solution and allowed to dry before the placement of a sterile fenestrated drape (iodine-based solutions can be used in patients with a history of chlorhexidine allergy). Equipment should be prepared on a sterile field and a sterile ultrasound probe cover used. At the end of the procedure the catheter insertion site should be covered with a sterile, transparent dressing.

Once in situ the catheter hubs should be cleaned with alcohol-based or chlorhexidine and alcohol wipes before and after the system is accessed and the line site should be

assessed daily for signs of infection. The need for central venous access should be regularly evaluated and the line removed if no longer required. Local protocols for CVC insertion and maintenance as well as staff education and monitoring of infection rates are all important components of minimising catheter-related blood stream infection.

4.1.2 Anatomy of the Extradural Space and Spinal Cord

Blood Supply – Matthew J Aldridge

Describe the arterial blood supply to the spinal cord.

The arterial supply to the spinal cord consists of the anterior and posterior spinal arteries. The single anterior spinal artery lies in the anterior median fissure of the cord. It is formed from branches of each vertebral artery at the level of the foramen magnum. It runs the whole length of the spinal cord. The anterior spinal artery supplies the anterior two-thirds of the spinal cord which is predominantly a motor area.

The posterior spinal arteries arise from the posterior inferior cerebellar artery at the level of the foramen magnum; they lie both anterior and posterior to the dorsal nerve roots. The two posterior spinal arteries supply the posterior third of the spinal cord (see Figure 4.1.2.1).

There are important contributions to the spinal arteries from radicular branches at each spinal level. The anatomy of this is variable. Commonly there is a dominant radicular branch to the anterior spinal artery called the great anterior radicular artery of Adamkiewicz (adam-ker-vits). Typically it arises from a low intercostal or a high lumbar artery, but sometimes the major contribution can arise from the iliac artery. This variation can lead to the development of anterior spinal artery syndrome; for example, following cross clamping of the aorta during aortic surgery.

Anterior spinal artery syndrome is flaccid paralysis at the level of ischaemia or infarction and a spastic paralysis with decreased pain and temperature appreciation

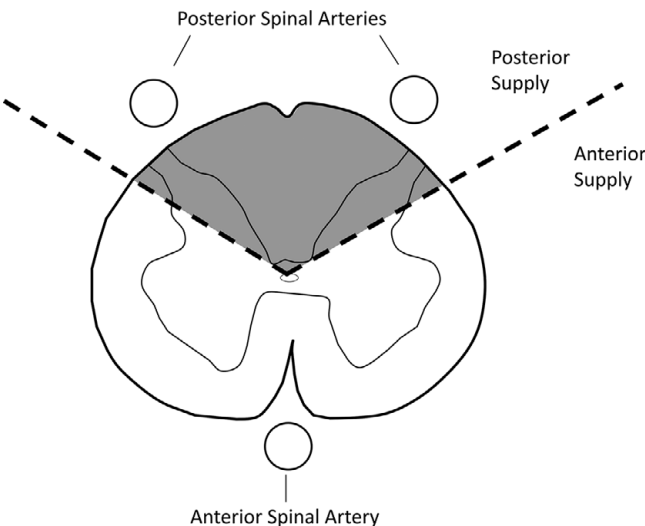


Figure 4.1.2.1 Blood supply to the spinal cord.

below the level of the lesion. There is relative sparing of the senses of proprioception and vibration, as the posterior columns are usually preserved.

What is the epidural or extradural space and what are its contents?

The epidural space is a continuous space within the vertebral column. It is the part of the spinal canal that is not occupied by the dural sac and its contents (i.e., the spinal cord!). It extends from the foramen magnum to the sacrococcygeal membrane at the lower end of the caudal canal.

The epidural space contains:

- Fat
- Lymphatics
- Loose connective tissue
- Nerve roots
- Arteries
- Venous plexus.

The pressure in the epidural space is usually found to be negative, particularly in the thoracic region. Although this may be related to stretching of the dural sac during extreme flexion of the back while performing an epidural.

What are the boundaries of the epidural space?

See Figure 4.1.2.2

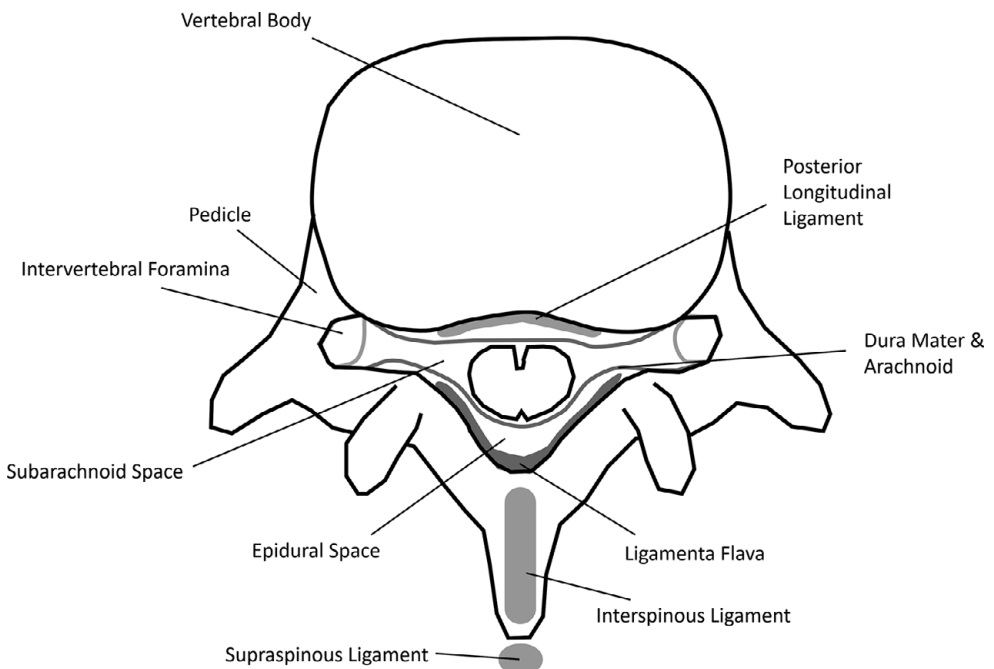


Figure 4.1.2.2 Anatomy of the epidural space.

The posterior boundary is the ligamentum flavum and the periostium of the laminae. The ligamenta flava connect adjacent laminae of each vertebra, and become progressively thicker in lower interspaces. The ligamenta flava may be paired at each level or be one pair of continuous longitudinal ligaments – opinion varies. Posterior to the ligamentum flavum is the interspinous then the supraspinous ligaments.

The anterior boundary is the bodies of each of the vertebrae and the intervertebral discs, which are covered by the posterior longitudinal ligament.

The lateral boundaries are the pedicles of the vertebral arches and the intervertebral foramina, which contain the nerve roots.

The superior boundary is the foramen magnum where the dura attaches to the cranium.

The inferior boundary is the sacral hiatus, which is covered by the sacrococcygeal membrane.

The spinal canal is triangular, with the base anteriorly. The space is deepest in the midline posteriorly. The epidural space is up to 3–4 times larger at the caudal as compared to the cephalad margin, so in cross section a saw tooth pattern will be seen. The epidural space is very thin anteriorly, but up to about 6 mm deep posteriorly in the lumbar region.

There has been debate surrounding the presence of a midline dorsal band of connective tissue called the plica mediana dorsalis, which was thought to draw the dura closer to the ligamentum flava in the midline, narrowing the space in the midline. The most recent conclusion is that this does not exist. However, there can be fibrous bands in the epidural space.

What are the important features of the venous system within the epidural space?

The veins of the epidural space run vertically but communicate freely. They are valveless and called the venous plexus of Batson. The plexus allows communication from the pelvic veins inferiorly to the cerebral venous system as well as to the azygos system. Air or drugs injected intravenously in the epidural space can therefore reach the brain and heart directly. Pelvic infection or malignancy spread can occur via Batson's plexus to the brain or vertebrae.

If thoracic or abdominal pressure is increased, the veins become relatively distended; thus the likelihood of intravascular placement of epidural catheter is increased, for example during a uterine contraction.

There is connection with abdominal and thoracic veins via the intervertebral foramina, which can transmit changes in venous pressure. Chronically increased intra-abdominal pressure or venous obstruction, such as inferior vena cava compression occurring due to the gravid uterus, can increase the calibre of veins, thus increasing the likelihood of venous placement of epidural needle or catheter. The increased vascular surface area may also increase absorption of local anaesthetics and therefore increase the likelihood of toxicity.

What can you tell me about the relevance of epidural fat?

The fat of the epidural space is different to most in the body. Firstly, obesity is unrelated to amount of epidural fat. Secondly, the quantity of epidural fat declines with age;

therefore elderly patients have a potentially larger epidural space. Finally, the fat is semi-liquid and contained within a capsule.

This is relevant as varying amounts of epidural fat may account for variation in local anaesthetic distribution and effect within the epidural space, as well as competing with neural tissue for local anaesthetic uptake.

What are the characteristics of a subdural anaesthetic block?

The subdural space is a potential space between arachnoid and dura mater. Local anaesthetic injection or catheter insertion into the subdural space may lead to an extensive and high sensory block with delayed onset despite negative aspiration. This may be mistaken for catheter migration or inadvertent subarachnoid block.

What techniques may be used to identify the epidural space during catheter insertion?

The 'loss of resistance' technique relies on sudden and marked loss of resistance to injection of saline or air on entering the epidural space. In addition, the 'hanging drop' has also been used to demonstrate sub-atmospheric pressure within the epidural space; however, this is considered less reliable due to variation in pressure at different levels of insertion. Ultrasound guidance can also be used to directly visualise needle insertion.

How can ultrasound be used to assist epidural catheter insertion?

Evidence suggests that ultrasound allows more accurate identification of vertebral level and needle insertion point than palpation of surface landmarks alone. This may reduce traumatic or failed attempts at insertion, and may be particularly useful with altered anatomy or when obesity makes palpation of landmarks more difficult. In addition, ultrasound may help predict the depth of the epidural space prior to needle insertion.

What is the paramedian approach to the epidural space?

This is an alternative to the commonly used midline approach and is useful in patients with altered anatomy or where positioning is difficult. The needle entry site is around 1–2 cm lateral to the midpoint of the spinous process immediately below the desired block level. The needle is inserted in a cephalad and medial direction until contact with lamina. At this point the needle is redirected cephalad and medially and walked off the lamina until it passes through the ligamentum flavum into the epidural space. Unlike the midline approach there is comparatively less 'feel' due to the absence of supraspinous or interspinous ligaments.

What are potential causes of headache in the postpartum period?

The most common causes have been found to be migraine or tension headache (47%), pre-eclampsia (24%) and post-dural puncture headache (16%). Other causes include cortical vein thrombosis, subarachnoid haemorrhage, posterior reversible leucoencephalopathy syndrome, space-occupying lesion, cerebral infarction, sinusitis or meningitis.

Due to significant and wide-ranging differentials a good history, physical examination and consideration of early neuroradiological imaging is essential.

What features are suggestive of a post-dural puncture headache?

This commonly occurs in the first 72 hours after dural puncture and presents as a frontal or occipital headache with a significant postural component. Severity increases on standing, coughing or straining and decreases on lying down.

Other symptoms include neck stiffness, nausea, vomiting, visual changes, hearing loss and tinnitus. In severe cases an abducens cranial nerve palsy may be present.

How should a post-dural puncture headache be managed?

Patients with suspected post-dural puncture headache should be referred to the anaesthetic team within 24 hours and followed up regularly until the headache resolves.

Conservative management has traditionally been the mainstay of management. Although bed rest may provide relief this should be balanced against the risk of venous thromboembolism. There is no evidence to support use of additional intravenous or oral fluid unless otherwise indicated due to dehydration.

Regular simple oral analgesia should be offered. There is limited evidence to support the use of caffeine. If used this should not exceed 24 hours of treatment with oral therapy, not exceeding doses of 300 mg (up to 900 mg in 24 hours), or a maximum of 200 mg in 24 hrs for women who are breastfeeding. There is no evidence to support use of other theophyllines, ACTH, steroids or triptans despite historical use. Acupuncture, greater occipital and sphenopalatine ganglion blocks also have insufficient evidence to recommend use.

Epidural blood patch should be considered when conservative therapy alone is ineffective, particularly where symptoms are causing difficulty performing activities of daily living or with care of the baby. Recent studies suggest complete relief of up to 1/3 of post-dural puncture headaches after a single epidural blood patch, with 50–80% describing at least partial relief. However, performing an epidural blood patch within 48 hours of dural puncture may be associated with lower success rates.

Further Reading

Macpherson D, Quondamatteo F, Broom M.

Update on applied epidural anatomy. *BJA*

Education. 2022; 22(5): 182–189.

4.1.3 Anatomy of the Trachea and Bronchi – Nirav D Patel and Mari H Roberts

This is most likely to come up in the science structured oral exam. Diagrams will help you remember the anatomy, which can be quite cumbersome to learn.

In your clinical practice, when have you used a flexible fibreoptic scope?

In my clinical practice I have used a flexible fibreoptic scope for awake and asleep fibreoptic intubations and to confirm placement of double lumen tube and bronchial blockers for one-lung ventilation. In the intensive care unit, I have seen flexible fibreoptic scopes used for bronchoscopy in ventilated patients.

Can you tell me about the anatomy of the trachea?

The trachea has a diameter of 1.5 to 2 cm and is 10 to 15 cm in length. It starts at the level of C6 and extends to form the carina at T4/5, where it divides into the left and right main bronchi. Anteriorly, it is made up of fibro-elastic connective tissue reinforced by 15 to 20 cartilaginous rings that are incomplete posteriorly. It is lined by ciliated columnar epithelium and mucus glands. The posterior part of the trachea is formed by smooth muscle which runs longitudinally and transversely.

What are the anatomical relations of the trachea at the C6 level?

You may want to draw a diagram or two to help you explain this (Figures 4.1.3.1, 4.1.3.2). It is useful to learn the cross-sectional anatomy at two levels: in the neck, usually at the level of C6 and in the thorax usually at the level of T4. The examiner may want the anatomy at both levels, or only at one. Practice drawing the diagrams and explaining the anatomy as you go along.

At the level of C6, the anterior relations of the trachea include the anterior jugular vein, sternohyoid muscle, sternothyroid muscle and thyroid gland. Lateral to the trachea, there are the sternocleidomastoid muscles, lobes of the thyroid gland and the carotid sheath, which contain the internal jugular vein, carotid artery, and vagus nerve. The oesophagus and recurrent laryngeal nerves are posterior to the trachea.

What about the anatomical relations of the trachea at the T4 level?

At the level of T4, the trachea is in the thorax. Anteriorly lies the manubrium, thymic remnants, and the inferior thyroid veins. Lower down, the brachiocephalic artery and left brachiocephalic vein cross the trachea anteriorly as well as the aortic arch. Lateral to both sides of the trachea lie the pleura, lungs, and vagus nerve. The superior vena cava and azygous vein are on the right of the trachea, and the aortic arch and left common carotid artery on the left. Posteriorly, lies the oesophagus and the left recurrent laryngeal nerve, which originates from the left vagus nerve as it crosses the aortic arch. The right recurrent laryngeal nerve is not present at this level as it originates from the vagus nerve as it crosses the subclavian artery, which is higher than the origin of the left recurrent laryngeal nerve.

What is the nerve supply to the trachea?

The nerve supply is from the recurrent laryngeal nerves and the sympathetic branches from the middle cervical ganglion.

During bronchoscopy, how would you identify that you are in the trachea and not further down in one of the bronchi?

Identification of the anterior tracheal rings and posterior trachealis muscle proximal to the bifurcation would confirm that I am in the trachea rather than further down into one of the bronchi.

Let's move down to the anatomy of the bronchi and bronchial tree.

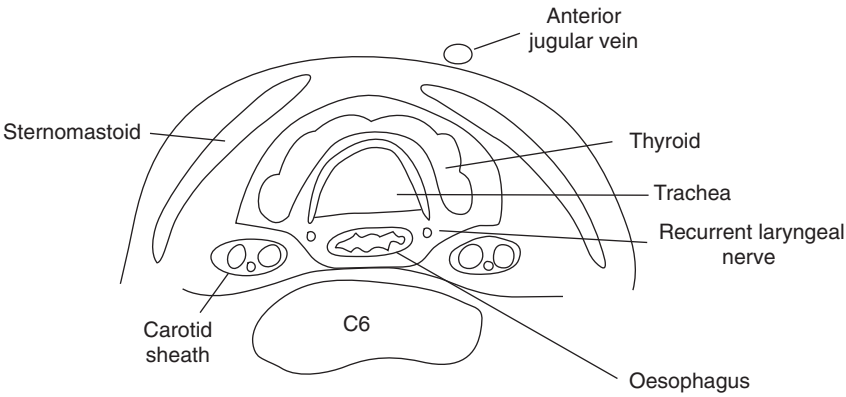


Figure 4.1.3.1 Cross section at C6.

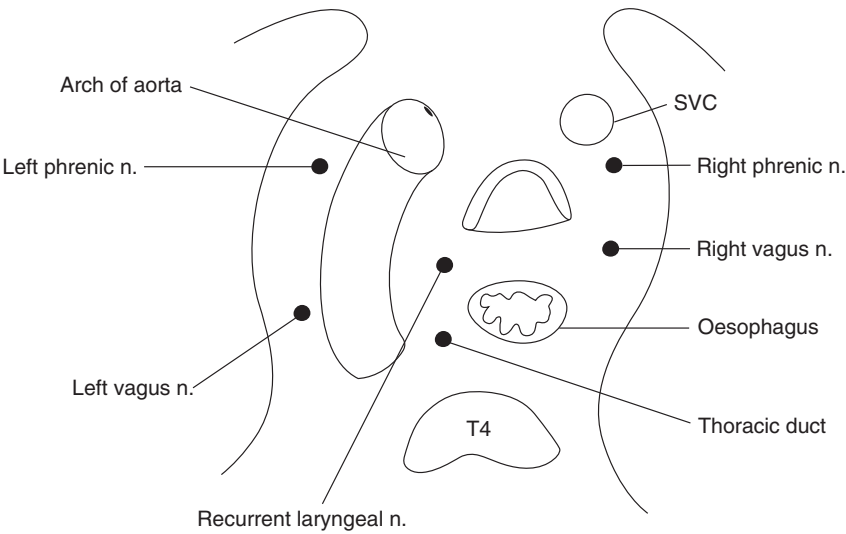


Figure 4.1.3.2 Cross section at T4.

What are the anatomical subdivisions of the bronchial tree from trachea to alveolus?

The subdivisions are bronchus, bronchioles, respiratory bronchioles, alveolar ducts, alveolar sacs and then alveolus.

What is the difference between the right and left main bronchus?

The right main bronchus comes off the trachea at 25 degrees and has a length of 2.5 to 3 cm. The left main bronchus comes off the trachea more horizontally with an angle of 45 degrees and is longer at about 5 cm. The left has a smaller diameter than the right bronchus.

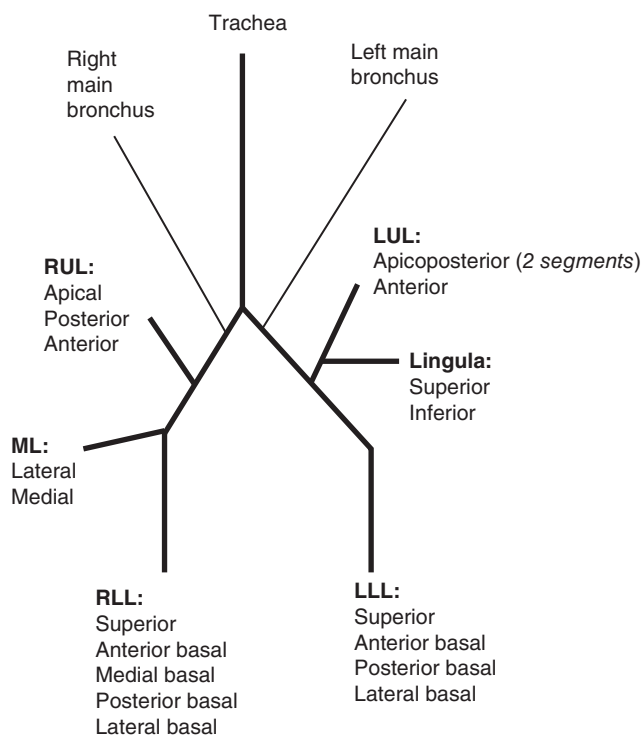


Figure 4.1.3.3 The bronchial tree and segments. Right lung contains ten segments and left lung contains nine segments.

How many bronchopulmonary segments are there in total and how do they differ between the right and left lung?

This is another opportunity to draw another diagram to help explain and name the various bronchopulmonary segments (Figure 4.1.3.3).

There are a total of 19 bronchopulmonary segments in both lungs, ten on the right and nine on the left. The right lung contains three lobes, the right upper, middle, and right lower lobe. The right upper lobe contains the apical, anterior, and posterior segments. The middle lobe divides into the lateral and medial segments. The right lower lobe divides into the superior, medial basal, anterior basal, lateral basal and posterior basal segments. The left lung gives rise to the left upper lobe and left lower lobe. The left upper lobe divides into apico-posterior segment (which is considered two segments), the anterior segment, and the lingula, which contains the superior and inferior segments. The left lower lobe divides into the superior, anterior basal, posterior basal and lateral basal segments.

Earlier on you mentioned double lumen tubes and one-lung ventilation. What are the indications for one-lung ventilation?

The indications for one-lung ventilation can be categorised into absolute indications and relative indications. Absolute indications include preventing damage or contamination of a healthy lung, for example from haemorrhage and abscess, controlling distribution of

ventilation in the presence of bronchopulmonary fistula, bullae, or bronchial trauma, and to facilitate single lung lavage. Relative indications include improving surgical access. Surgeries that are very likely to require one-lung ventilation include thoracic aneurysm repair, VATs, pneumonectomy, lobectomy, minimally invasive cardiac surgery, oesophagectomy, and mediastinal mass reduction.

What options are available to achieve one-lung ventilation?

Options to achieve one-lung ventilation include double lumen tubes, bronchial blockers and advancing a single lumen tube into the bronchus of the non-operative or healthy lung. The last option is reserved for emergencies or difficult airways.

What size double lumen tubes are available?

Double lumen tubes are available as left or right sided. They are available in 35, 37, 39 and 41 French gauge. A 37 French is usually suitable for an average-size woman and 39 French for a man.

You are anaesthetising a patient for a left lower lobectomy – would you use a left- or a right-sided double lumen tube?

I would use a left-sided double lumen tube because this is suitable for most procedures. There is a risk of occluding the right upper lobe using a right-sided double lumen tube as it comes off within about 2.5 cm from the origin of the right main bronchus

How would you insert a double lumen tube?

Prior to induction of anaesthesia, I would check both the integrity of the tracheal and bronchial cuffs, lubricate the outside of the double lumen tube and insert a pre-shaped stylet. After inducing anaesthesia and allowing adequate muscle relaxation, I would perform laryngoscopy to visualise the glottis. The double lumen tube is initially inserted with the bronchial curve facing anteriorly. Once the bronchial tip is through the larynx I would ask my assistant to remove the stylet. The tube is then rotated 90 degrees to the corresponding side (anticlockwise for left-sided and clockwise for right-sided). It is then advanced beyond the glottis until resistance is felt. The double lumen tube is then connected to the breathing circuit using a double catheter mount and the tracheal cuff is inflated until the air leak is stopped. I would check for air entry; CO₂ trace and temporarily secure the double lumen tube. Generally, depth of insertion can be given by the formula $12 + (\text{patients height}/10)$ measured at the teeth but I would still perform checks to ensure correct placement.

How would you confirm placement of the double lumen tube?

After inflating the tracheal cuff, I would inspect the chest for bilateral chest wall movement and auscultate both sides of the chest for air entry. I would check that there is an adequate CO₂ trace. The next step is to check for adequate isolation of each lung starting with the bronchial lumen. This would be done by clamping the tracheal limb of the breathing circuit and disconnecting it from the tracheal lumen of the double lumen tube. The bronchial cuff would be inflated, and ventilation will only continue through the bronchial lumen. I would auscultate the chest to confirm unilateral air entry and no air leak. After this, the tracheal lumen of the double lumen tube would be reconnected to the

breathing circuit and clamp removed to restart two-lung ventilation. Isolation of the other lung would now be tested. The bronchial lumen would be clamped, and bronchial lumen disconnected from the breathing circuit. Ventilation is now only occurring through the tracheal lumen. Unilateral air entry on auscultation would confirm successful isolation. The bronchial lumen would be reconnected to the breathing circuit, clamp removed, and two-lung ventilation restarted. The final check for placement would be to use a flexible bronchoscope. I would pass a fibrescope through the tracheal lumen and identify the carina and the bronchial cuff as a blue moon crescent in the left main bronchus but not herniating over the carina after inflation. I would also visualise the right upper lobe to confirm the tracheal lumen sits in the trachea. After performing the checks, I would deflate the bronchial cuff and secure the double lumen tube. The checks would be repeated if the patient is then placed into the lateral position as the double lumen tube can become displaced.

When would you have to use a right-sided double lumen tube and what additional checks would you have to perform other than those mentioned above?

Indication for a right-sided double lumen tube include surgery involving the left main bronchus such as left pneumonectomy, left lung transplant or if there is distortion of the left main bronchus by a tumour or aneurysm. The same checks as mentioned before would be performed but in addition to this, a fiberoptic bronchoscope would be passed into the bronchial lumen to ensure the Murphy's eye of the bronchial lumen is aligned with the right upper lobe.

Further Reading

Ashok V, Francis J. A practical approach to one-lung ventilation. *BJA Education*. 2018; 18 (3):69–74.

4.1.4 The Anatomy of the Autonomic Nervous System – Charlotte Morris, Daniel El-Dalil and Gareth J Gibbon

This is a frequently examined topic. Take your time because it is very easy to tie yourself up in knots!

Tell me what you know about the autonomic nervous system (ANS).

Start generally. Define and classify and try to be systematic in your answer.

The autonomic nervous system is made up of the nerves and humoral systems that maintain homeostasis in the body and is not under conscious control. There is constant activity from both the sympathetic and parasympathetic components. These differ anatomically, pharmacologically and physiologically. The ANS is controlled by centres in the hypothalamus, medulla and brainstem. The autonomic nerves, unlike the somatic nervous system, leave the central nervous system and synapse before reaching the effector organ. Myelinated first order nerves synapse on to unmyelinated second order nerves, which in turn supply the effector organ. Sympathetic nerves originate in T1–L2 segment of the spinal cord. The parasympathetic neurones arise from cell bodies in the

communicantes. Usually they synapse here onto second order grey rami communicantes. These are unmyelinated fibres which can then run back towards the spinal nerve and follow its course.

Is there anything else that can happen to these neurones once they leave the spinal cord?

There are three possible fates of the first order neurones:

1. They can synapse in the chain before running back to follow the path of their originating spinal nerve.
2. They can run up or down the sympathetic chain to synapse in a more distant ganglion within the chain.
3. They can pass without synapse through the chain towards a peripheral ganglion. This latter course occurs with the sympathetic supply to the abdominal and pelvic organs. These preganglionic sympathetic fibres run in the splanchnic nerves. The greater splanchnic nerve arises from the T5 to T9 segments of the sympathetic chain. The lesser splanchnic nerve runs from the T10 and T11 segments. Sometimes there is a third least splanchnic nerve from T12. All supply the coeliac plexus, which is where they synapse on to unmyelinated nerves. After they have synapsed, all the sympathetic second order unmyelinated nerves tend to follow the arterial supply to their effector organs.

What about the parasympathetic nervous system – how does that differ from the sympathetic nervous system?

The parasympathetic nervous system differs anatomically, pharmacologically and physiologically. Anatomically the parasympathetic efferents leave the CNS to synapse onto second order neurones close to the target organ. There is a cranial component and a sacral component. The cranial component is conveyed in cranial nerves III, VII, IX and X from specific nuclei in the brainstem. These follow the course of the respective cranial nerves to synapse at four ganglia. The sacral component efferents are termed the nervi erigentes and arise from S2, S3 and S4. These join the pelvic plexuses and are distributed to the pelvic organs. Small ganglia in the visceral walls relay the postganglionic fibres. Pharmacologically, the neurotransmitter is acetylcholine which acts on muscarinic receptors at both the ganglion and effector organs. Physiologically the parasympathetic system antagonises the effects of the fight or flight sympathetic system. It causes constriction of the pupils, bradycardia, bronchoconstriction, mucus production, peristalsis and stimulates the production of pancreatic and gastric secretions. The nervi erigentes are nerves of emptying and controlling bladder and bowel function.

How is the adrenal gland innervated?

The adrenal innervation is different. White myelinated fibres pass through the coeliac ganglion towards the adrenal gland and innervate the adrenal medulla. Here they release acetylcholine to stimulate the secretion of adrenaline and noradrenaline from the medulla. Embryologically the adrenal medulla and the sympathetic nerves share a common origin.

You have mentioned the coeliac plexus. Do you know of any other plexuses?

Yes. All the thoracic and abdominal organs are innervated via plexuses. These plexuses are simply clusters of post-ganglionic nerves. There is a cardiac plexus supplied from T1 to T4. The cardiac plexus supplies the heart. It also supplies the lungs via a pulmonary plexus. In the abdomen, there is an aortic plexus formed from the post-ganglionic fibres of the coeliac plexus. This supplies further plexuses, named after the organs which they supply, again following the blood supply. The hypogastric plexus forms from the aortic plexus and the lumbar sympathetic trunks. This feeds right and left pelvic plexuses and the pelvic organs.

Can you tell me more about the coeliac plexus?

This is the largest of the sympathetic plexuses and is at the level L1, originating where the coeliac artery branches off the aorta. It acts as a conduit for the sympathetic supply to most of the abdominal organs. It can be blocked to stop pain pathways from the pancreas and upper GI tract.

What are the indications and potential complications of a lumbar sympathectomy?

It would be fair for the examiner to ask about any blocks you have spoken about in more detail. Always have a systematic approach to discussing any procedure –it ensures you don't miss out on any easy marks.

The aim of a lumbar sympathectomy is to block the distal lumbar sympathetic outflow as well as impair any conduction through afferent pain fibres that can relay through the sympathetic chain. Local anaesthetic can be used for short-lasting effect and phenol (typically 2% or 3%) or alcohol can be used if a longer duration of symptomatic treatment is felt appropriate. There are indications, contraindications and complications. Indications include painful lower limb ischaemia, complex regional pain syndromes, phantom pain and frostbite to the lower limb. Contraindications, as with most procedures, would be infection at the site of instrumentation, patient refusal, allergy to any of the agents to be used, tumour involvement at the site of entry and coagulopathy. Complications would include those caused by the procedure, and those resulting from the procedure. There might be damage to and bleeding from intercostal vessels, the aorta or inferior vena cava. Local haematoma and infection might also complicate the procedure. As a result of the injection, the ipsilateral leg will become subjectively and objectively warmer and more erythematous. Post-sympathectomy neuralgia occurs commonly manifesting as pain in the thigh or groin. There is also the potential for sexual dysfunction problems, especially if the block is bilateral. There is always the risk that the block fails.

How would you perform a lumbar sympathetic block?

As with every procedure I would see the patient on the ward, ensure that the potential benefit of the procedure outweighed the risk, establish the absence of contraindications and ensure that full consent has been ascertained. I would perform a lumbar sympathectomy under skilled supervision as I have never attempted this procedure before. I would ensure trained anaesthetic staff were present, that my emergency drugs were

drawn up and full monitoring had been applied. I would consider the need for sedation and discuss this with the patient prior to the procedure. I would maintain strict asepsis. As with the coeliac plexus block, this procedure is usually performed under ultrasound or fluoroscopic guidance, with the patient in the prone position. Once the correct anatomy has been identified I would clean the area to maintain asepsis. The L2 or L3 vertebra is identified and after infiltration of local anaesthetic to the skin, a needle inserted 10–12 cm from the midline. The needle is aimed towards the anterolateral border of the body of L2 or L3. Radio-opaque contrast can be injected to confirm that the injection will be made in the correct tissue plane. After confirmation of position, aspiration and contrast can proceed. I understand that neurolytic blocks are made more effective by repeating the procedure at the L3 and L4 vertebrae.

Do you know where the ganglia are for the cranial nerve outflow?

The third nerve transmits fibres from the Edinger-Westphal nucleus towards the ciliary ganglion in the orbit. Stimulation leads to pupillary constriction. The seventh nerve transmits fibres from the superior salivatory nucleus to two ganglia. One is via the greater petrosal nerve to the pterygopalatine ganglion in the pterygopalatine fossa which then runs on to supply the lacrimal glands. The other is via the chorda tympani to the secretomotor supply to the salivary glands through the submandibular ganglion. The submandibular ganglion is closely related to the lingual nerve in the floor of the mouth. The ninth nerve transmits fibres from the inferior salivatory nucleus to the otic ganglion via the lesser petrosal nerve. The otic ganglion lies below the foramen ovale. It supplies secretomotor fibres to the parotid gland.

And the vagus?

The vagus conveys the most widely distributed parasympathetic supply. The parasympathetic neurons arise from the dorsal nucleus in the medulla oblongata. They are carried with the vagus nerve and distributed through the cardiac, pulmonary and other organ plexuses. Again, the first order myelinated nerves pass through these plexuses to small ganglia in the walls of the target viscera. It is responsible for parasympathetic autonomic tone in all abdominal and thoracic viscera from the neck to the transverse colon. The majority of the neurones running in the vagus are afferent, conveying sensory information from the viscera. Significantly for anaesthetists it controls the motor and sensory supply to the larynx and vocal cords.

Further Reading

Alexander CE, Jesus OD, Varacallo M. Lumbar sympathetic block. *StatPearls*. 2022.

D'Souza RS, Warner NS. Phenol nerve block. *StatPearls*. 2022.

Smith G, Aitkenhead AR. *Smith and Aitkenhead's Textbook of Anaesthesia*. 7th ed Jonathan Thompson Iain Moppett, Matthew Wiles (eds). Elsevier; 2019.

4.1.5 Cranial Nerve Anatomy – Poonam Bopanna

The anatomy of the cranial nerves is rather dry and cumbersome to remember. The following podcast aims to give a concise description of the main cranial nerves relevant to anaesthetists.

Describe the anatomy of the optic nerve.

The optic nerve is the second cranial nerve (CN II) and is the sensory nerve to the retina. Its fibres originate from the innermost layer of the retina called the stratum opticum. These fibres are axons of the cells in the retina and pierce the sclera to form the optic nerve. It is surrounded by the cranial meninges containing an extension of the subarachnoid space. The optic nerve runs through the optic canal and enters the cranial cavity where it joins with the nerve from the other side, forming the optic chiasma. In the optic chiasma the fibres from the nasal half of the retina cross the median plane and enter the optic tract of the opposite side. However, the temporal fibres remain and enter the optic tract on the same side. The decussation of the nerve fibres in the chiasma means that the right optic tract conveys impulses from the left visual field while the left optic tract conveys impulses from the right visual field. The optic tracts then continue backwards to terminate in the lateral geniculate body of the thalamus. From these nuclei axons are relayed to the visual cortices of the occipital lobe of the brain.

Tell me about the motor nerve supply to the eye?

The third cranial nerve (CN III) is the oculomotor nerve and is the motor supply to all the extraocular muscles except the superior oblique and the lateral rectus. In addition, it also carries parasympathetic supply to the sphincter pupillae and the ciliary muscles. The nerve arises in upper midbrain, where the nuclei lie in the periaqueductal grey matter. Nerve fibres then pass between the cerebral peduncles, pierce the dura mater and run in the lateral wall of the cavernous sinus. CN III exits the skull via the superior orbital fissure to enter the orbit. Within the superior orbital fissure, it divides into the superior branch that supplies the superior rectus and levator palpebrae superioris and the inferior division that supplies the medial rectus, inferior rectus and the inferior oblique.

The trochlear nerve (CN IV) supplies the superior oblique muscle. It arises from a nucleus in the periaqueductal grey matter of the lower midbrain. Fibres pass through the posterior cranial fossa following the edge of the tentorium just lateral to CN III. Next the fibres pass through the middle cranial fossa in the lateral wall of the cavernous sinus before entering the orbit through the superior orbital fissure.

The abducent nerve (CN VI) supplies the lateral rectus muscle. The nucleus of the abducent nerve lies in the inferior pons. Fibres then pass through the posterior and middle cranial fossa, through the cavernous sinus and into the orbit through the superior orbital fissure. CN VI has a very long intracranial course and is susceptible to damage by stretching in patients with raised intracranial pressure.

What are the clinical signs of a third nerve palsy?

The clinical signs of a complete nerve palsy include:

- Ptosis
- Loss of pupillary light reflexes
- Dilatation of the pupil
- Downward and outward gaze due to unopposed action of the superior oblique and lateral rectus muscles
- Loss of accommodation.

What are the symptoms of a fourth cranial nerve palsy?

With a fourth nerve palsy the patient will experience diplopia on looking downwards because the eye is pulled down only by the inferior rectus and therefore moves slightly differently to the other side.

Outline the anatomy of the trigeminal nerve?

The trigeminal nerve (CN V) is the fifth cranial nerve and provides sensory supply to the face, nasopharynx, nasal and oral cavities, paranasal air sinuses and the anterior part of the scalp. Its motor branch supplies the muscles of mastication.

It has three sensory and one motor nucleus. The three sensory nuclei are:

- The **principle sensory nucleus** which lies in the upper pons and receives fibres transmitting touch sensation.
- The **mesencephalic nucleus** which lies in the midbrain and receives proprioceptive fibres.
- The **nucleus of the spinal tract** of the trigeminal nerve which lies deep to a tract of descending fibres running from the pons to the substantia gelatinosa of the spinal cord. This nucleus receives pain and temperature nerve input.

The motor nucleus is situated in the upper part of the pons and lies just medial to the principal sensory nucleus.

The nerves that originate from the above nuclei pass forward to the trigeminal ganglion (also called the semilunar ganglion due to its shape), which lies near to the apex of the petrous temporal bone. The sensory fibres pass through the ganglion whereas the motor fibres pass below the ganglion. The three main sensory divisions of the nerve emerge from the anterior border of the trigeminal ganglion and are the ophthalmic division (V1), the maxillary division (V2) and the mandibular division (V3).

The ophthalmic division (V1) of the trigeminal nerve divides into the lacrimal, nasociliary and frontal branches just before the superior orbital fissure. The frontal nerve further divides into the supraorbital and supratrochlear nerves. V1 also transmits some sympathetic and parasympathetic fibres. The ophthalmic division supplies the skin of the nose, the forehead, eyelids, scalp, conjunctiva, lacrimal apparatus and the globe.

The maxillary division (V2) runs below the ophthalmic division and leaves the base of the skull via the foramen rotundum. After traversing the pterygopalatine fossa, it becomes known as the infraorbital nerve which passes through the infraorbital foramen to supply skin to the surrounding areas of face.

The mandibular division (V3) also carries the motor fibres the trigeminal nerve. It leaves the skull via the foramen ovale and subsequently divides into sensory and motor branches. The motor root supplies the muscles of mastication. The sensory branches are the meningeal, buccal, auriculotemporal, inferior alveolar and lingual. These branches supply the lower third of the face and floor of the mouth.

Tell me about the facial nerve?

The facial nerve is the seventh cranial nerve (CN VII) and carries:

- Visceral motor fibres supplying the facial muscles of expression
- Visceral sensory fibres provide input from taste sensation from the anterior two-thirds of the tongue

- Visceral parasympathetic fibres are secretomotor to the salivary glands and lacrimal gland

The facial nerve leaves the pons and passes through the internal auditory meatus to the facial ganglion. At the facial ganglion the parasympathetic fibres leave the rest of the facial nerve and travel in the greater petrosal nerve to the pterygopalatine ganglion. The remaining facial nerve turns posteriorly and descends through the bony canal in the posterior wall of the middle ear then emerges through the stylomastoid foramen. Just above the stylomastoid foramen the chorda tympani nerve arises, which joins with the lingual nerve to convey taste from the anterior tongue, parasympathetic innervation for the submandibular gland and some motor fibres. The remaining facial nerve has only motor actions and as it passes through the parotid gland it divides into the temporal, zygomatic, buccal, mandibular and cervical branches. These branches supply the muscles of facial expression, buccinators and platysma.

What does the vagus nerve innervate?

The vagus nerve has numerous components:

- Parasympathetic fibres to the heart, lungs and alimentary canal
- Sensory fibres from the heart, lungs and alimentary canal
- Motor fibres to the larynx, pharynx and palate
- Sensory fibres from the larynx, pharynx and palate
- Somatic sensory fibres from the external acoustic meatus and tympanic membrane.

Describe the course of the vagus nerve.

The vagus nerve (CN X) has the most extensive course compared to any other cranial nerve. It emerges from the medulla and exits the skull via the jugular foramen, alongside the glossopharyngeal nerve, accessory nerve and internal jugular vein. The vagus nerve descends through the neck invested in the carotid sheath and lying between the internal jugular vein and internal and common carotid arteries. In the root of the neck the right vagus nerve descends in front of the subclavian artery to enter the thorax, while the left vagus nerve descends between the common carotid and left subclavian arteries. The two vagus nerves then pass posteriorly to each main bronchus to form the pulmonary plexus. Next, they converge onto the oesophagus to create the oesophageal plexus. The anterior and posterior vagus nerves emerge from the oesophageal plexus and travel asymmetrically through the thorax and abdomen to supply the alimentary tract.

What do the 9th, 11th and 12th cranial nerves innervate?

The ninth cranial nerve (CN IX) is the glossopharyngeal nerve and has both general and visceral sensory, motor and parasympathetic fibres. The general sensory fibres supply the middle ear, pharynx, posterior two-thirds of the tongue and the carotid sinus and carotid body. The visceral sensory fibres carry taste sensation from the posterior third of the tongue. The parasympathetic fibres supply the parotid gland. The motor fibres supply stylopharyngeus. To test the integrity of CN IX a gag reflex can be tested. It will be absent on the side of the lesion.

The accessory nerve (CN XI) supplies the sternocleidomastoid and trapezius muscles resulting in a weakened shrugging of the shoulders and rotation of the neck.

The hypoglossal nerve (CN XII) supplies all the intrinsic and extrinsic muscles of the tongue except palatoglossus. It can be injured during tonsillectomy and results in ipsilateral paralysis of the tongue. Thus, when the tongue is protruded, it will deviate to the side of the lesion.

Further Reading

Craven J. The cranial nerves. *Anaesthesia and Intensive Care Medicine*. 2007; 8 (12): 499–503.

Moore KL, Agur AMR. *Essential Clinical Anatomy*. Second edition. 2002. Lippincott Williams & Williams.

4.1.6 Anatomy of the Intrathecal Space – Alexandra K Freeman

What can you tell me about the intrathecal space?

The brain and spinal cord are covered by three protective membranes: dura mater (the outermost layer), arachnoid mater (the middle layer) and pia mater (the innermost layer). The intrathecal, or subarachnoid, space is situated between the arachnoid and pia mater. These two membranes are joined by loose connective tissue trabeculae which form a delicate mesh within the intrathecal space, through which CSF flows freely.

Throughout the cranial vault and spinal column, the depth of the subarachnoid space has considerable variability. Areas where the arachnoid and pia mater diverge, creating vaults of CSF, are called subarachnoid cisterns. The CSF has multiple functions to protect and support the central nervous system; it acts as a shock absorber, delivers nutrients and removes waste products.

The intrathecal space runs from the cranial vault to the level of S2. It can be anatomically divided into the cranial intrathecal space and the spinal intrathecal space. It is the spinal intrathecal space which we access when performing a spinal anaesthetic or lumbar puncture.

What are the contents of the intrathecal space?

Contained within the intrathecal space are:

- A network of connective tissue trabeculae
- Free flowing CSF
- Cranial and spinal nerves
- Blood vessels.

List the structures passed through during performance of a spinal block in the midline position.

The layers passed through, from superficial to deep, are:

- Skin and subcutaneous tissue
- Supraspinous ligament
- Interspinous ligament
- Ligamentum flavum

- Dura mater
- Arachnoid mater.

Where does the spinal cord end? Why is this important?

The spinal cord terminates at the conus medullaris, the level of which varies with age. At birth, the conus medullaris lies approximately at the level of L3. In the adult, the conus medullaris lies approximately at the level of L1. There is, however, variability within the population and in up to 50% of adults the conus medullaris lies below L1. This is important to consider during a spinal anaesthetic or lumbar puncture as there is a risk of damage to the spinal cord by the needle.

Which factors determine the level of a spinal block? How can these be modified during performance of the block?

This question specifically relates to block height. It may also be useful to refresh your knowledge on other pharmacokinetic variables of local anaesthetic solutions.

The factors can be divided into those related to the local anaesthetic solution (which are mostly modifiable) and those related to the patient (which are mostly non-modifiable).

Local anaesthetic factors include:

- Baricity
- Volume and dose of local anaesthetic
- Level of injection.

Baricity of local anaesthetic solutions determines density and, therefore, movement through the CSF with gravity. Hyperbaric solutions (which may be achieved by adding glucose) are denser than CSF and, therefore, move downwards with gravity; the converse is true for hypobaric solutions.

Volume of local anaesthetic affects block height, particularly when hyperbaric solutions are used. Larger volumes provide anaesthesia for a greater number of dermatomes. Dose of local anaesthetic is usually a consequence of altering volume but also has an effect on duration of anaesthesia.

Level of injection has also been shown to affect block height but is limited anatomically due to termination of the spinal cord (cranially) and the sacrum (caudally).

Patient factors include:

- Patient position
- Volume of CSF
- Patient height
- Raised intra-abdominal pressure.

Patient positioning can be used to manipulate the spread of hyper- and hypo-baric solutions within the intrathecal space. For example, sitting a patient up can facilitate a dense sacral block or lying the patient in the lateral position can facilitate a unilateral block.

The volume of CSF is approximately 150 ml, distributed between the cranium and spinal column. Patient height and factors causing raised intra-abdominal pressure (for example pregnancy or obesity) may alter the volume of spinal CSF and, therefore, the spread of local anaesthetic solutions. Modification of local anaesthetic delivery based on these factors must be done with caution, however, as the effects are not reliable.

How would you assess adequacy of a spinal block prior to a lower segment caesarean section?

I would assess three components: motor block, sensory block to cold and sensory block to light touch. I would assess motor block using the Bromage scale, expecting a dense motor block of both lower limbs. I would assess sensory block to cold using an ethyl chloride spray, expecting a block from S5 to T4 bilaterally. I would assess light touch using a cotton wool ball, expecting a block from S5 to T5 bilaterally.

Describe your management if the block was too low.

Always remember that 'management' does not only refer to your medical interventions. You must demonstrate situational awareness, including communication with the theatre team and your supervising Consultant, counselling of the patient, careful documentation and arranging appropriate follow-up.

My management would depend upon the timing at which the inadequacy of the block was identified, the urgency of delivery of the baby and any additional patient factors. In all cases I would prioritise the comfort and safety of the mother.

If the low block was identified prior to skin incision and delivery of the baby was time critical, provided there were no absolute contraindications, I would offer a general anaesthetic. I would ensure judicious vasopressor administration to mitigate the confounding vasodilatory effects of the spinal block and the anaesthetic agents.

If the low block was identified prior to skin incision but the surgery was not time critical, I would first attempt to increase the block height through repositioning the patient, either by increasing the Trendelenberg tilt or by flexing the patient's hips and knees (in order to reduce the lumbar lordosis). If this did not provide adequate improvement, the options would be to delay the procedure until the block had worn off and could be repeated, insert an epidural catheter and perform a combined spinal and epidural, or perform a general anaesthetic. I would discuss these options with the surgical team, my supervising consultant and the patient.

If the patient complained of pain during the procedure, I would ask the surgeons to stop and carefully assess the block. I would reassure the patient and offer supplementary analgesia, in the form of Entonox or intravenous opioids (e.g. 100 mcg boluses of alfentanil, titrated to effect). If appropriate, for example during closure of the incision, I could ask the surgeons to infiltrate local anaesthetic. If these interventions did not provide a prompt resolution of the pain I would offer the patient a conversion to general anaesthetic.

In all cases I would carefully document the events, including all interventions offered, and arrange for the patient to be reviewed postoperatively.

4.1.7 Trunk Blocks – Farzad Saadat and Sarah F Bell

Questions around trunk blocks could lead to simple anatomy questions or wider questions around acute pain management

You are called by the thoracic team on the ward to help with the pain management of a 67-year-old man who fell from a 2 m ladder about 12 hours ago. On admission, he had a head to pelvis CT scan, which showed left-sided posterior fractures of ribs 2–8, with anterior fractures of 3–5, constituting a flail segment. His PMH includes

hypertension and AF, for which he takes ramipril and Apixaban. He last took the Apixaban 24 hours ago.

The patient is on the thoracic ward having been admitted via A&E and has been prescribed regular co-codamol by the trauma team. Let's start by thinking about what might be your concerns with this patient.

My concerns can be categorised into those related to the recently sustained injuries, the risk of secondary complications from the fractures, and the patient's pain control. In terms of his rib fractures, the patient is at risk of lung injury such as pneumothorax, contusion or haemothorax, particularly given his apixaban. I would want to ensure he had been screened for any other injuries given his mechanism of injury and to perform a secondary survey if it had not already been done, as well as check the full report of his CT scan. The patient is also at risk of developing pneumonia, particularly if his pain control is inadequate and he is struggling to breathe. Therefore, his analgesia needs to be optimised as a priority.

How would you assess this patient's pain needs? Do you know any scoring systems that can help you?

My assessment would include a full A to E assessment as mentioned, as well as using a scoring system such as the Chest Trauma Score (CTS) or STUMBL score, which calculates the patient's risk of secondary complication. In addition, I would assess the efficacy of any analgesia that has been given to this point, as it will help guide further decisions.

The STUMBL score uses the number of rib fractures, the patient's age, the patient's oxygen requirement, the presence of a flail segment, any anticoagulants taken and chronic lung disease to risk stratify patient. This patient would score over 30, even without knowing his oxygen requirement, which would put him in the most high-risk category, and I would urgently seek to get his pain under control.

There are many pain scoring systems, but my primary concerns with this patient would be to establish if he can deep breathe, cough and mobilise comfortably. These will be my markers of success for any analgesic intervention.

The patient is unable to take deep breaths and refuses to cough due to pain. How would you manage this patient?

My management would involve supportive therapy, analgesia and deciding where best to manage this patient. For supportive therapy, the patient may require supplemental oxygen, aiming for SpO₂ of at least 94% and physiotherapy when his pain is better controlled.

Options for analgesia for rib fractures include analgesics on the WHO pain ladder, lidocaine patches, IV opioids given via a PCA and/or regional anaesthesia. Given the high risk of secondary complication in this case, and the inadequacy of his analgesia to this point, I would arrange for regional anaesthesia as soon as possible. I would optimise his analgesia while the regional block is being prepared by adding NSAIDs if they are not contraindicated, a long-acting opiate such as MST, oramorph for breakthrough pain and consider a lidocaine patch.

I would discuss with his thoracic team where his ongoing management should be; he is at very high risk of complications and may benefit from being nursed in an HDU setting.

What are your options for regional anaesthesia in this case?

The most commonly performed procedures for analgesia for rib fractures are epidural, paravertebral block, serratus anterior block and erector spinae block (ESP). An epidural is not appropriate in this case, given he last took apixaban 24 hours ago and this needs to be stopped for a minimum of 48 hours for safe epidural placement. Since the patient has unilateral fractures, serratus anterior, an ESP or paravertebral catheter technique would be appropriate in this case.

Describe how you would perform your block of choice.

It can be daunting to remember the procedure for different regional blocks; however, most of the logistics are the same for almost all of them. Think about how you would set up for any regional anaesthesia procedure.

I would perform an ESP block. I would consent the patient prior to the procedure and organise the location and logistics, equipment, and drugs for injection and for emergency use prior to starting. If possible, I would perform the procedure in theatre or an anaesthetic room with the help of an ODP. Before getting scrubbed, I would ensure that the patient had ECG, saturation and blood pressure monitoring and oxygen if required. I would assess whether any additional analgesia was required to help optimise the patient's position for the block and I would ensure that the patient had IV access. This is an aseptic procedure, and so I would scrub and set up a sterile field, using a sterile probe cover on the ultrasound probe. I would use an echogenic regional needle and have an experienced assistant available to assist me. If possible, I would have the patient in a sitting position. I would set up the ultrasound so I can clearly see it without turning my head.

Prior to starting the procedure I would perform a 'stop before you block' safety check. The procedure requires clear communication with the patient throughout. I would clean the skin with 2% chlorhexidine swabs and aim to perform the block at T5, as this would provide good spread above and below the injection point. I would place my transducer in a paramedian sagittal orientation at T5, identify key structures, then use 1% lidocaine to topicalise the skin and point my needle in a cephalad to caudad direction as I perform the block.

Which ultrasound probe would you use and why?

The choice of ultrasound probe depends on the distance of target structures. Generally, the higher frequency, linear probes can be better used to image relatively superficial structures, up to 5–6 cm away. By contrast, the low frequency curvilinear probe is better for further target objects, more than 5 cm away. In most ESP blocks, a high frequency linear probe can be used; however, if the patient is of large body habitus, the curvilinear probe may be required.

Tell me about the choice of local anaesthetic.

An ESP block is a 'plane block' and so it requires a large volume of local anaesthetic to open the space. I would aim to inject 40 ml of either 0.25% bupivacaine or 0.2%

ropivacaine, provided this does not exceed the maximum dose for this patient. The lower concentration would allow the high volume block to be administered, while giving good relief of pain for 8–10 hours.

Describe the relevant anatomy when performing this block, and how you would administer the block.

Anatomy questions are fair game in your final viva; the basic anatomy relevant to a block is on the syllabus. Being able to discuss the anatomy in terms of how you see it on an ultrasound screen shows knowledge and demonstrates experience with the procedure.

I would place the transducer in a paramedian, sagittal orientation at the level of T5, around 2 cm lateral to the spinous process. I would look for the shadow of the transverse process of T5 and the three layers of muscle above it, which are, from superficial to deep, trapezius, rhomboid and erector spina. Above and below each transverse process is a paravertebral space. The transverse process has a squared appearance on its top, with classic bony acoustic shadowing. If the structure looks too rounded, my probe may be too lateral and I may be seeing a rib, rather than a transverse process.

I would use an in-plane technique, inserting the needle so that it is pointing from a cranial to a caudal direction, aiming for the transverse process of T5. I would aim to keep my needle visible at all times and should feel bone when I reach the transverse process. When in the correct position, I would give a test injection of 3–4 ml and look for lifting of the ESP needle away from the transverse process. If it appears I am in the correct space, I would inject the rest of the 40 ml of local anaesthetic. I should see the space under the ESP continue to expand, and see the same happening on the transverse processes above and below T5.

How would you ensure ongoing pain management?

Levobupivacaine has an elimination half-life of 210 minutes. I would expect a single-shot ESP block of this kind to last 8–10 hours. For ongoing pain relief I would place a catheter with an ongoing infusion device instead of a single-shot block technique. This would prevent the need for repeated procedures for this patient. Important considerations when placing a catheter include a more complex insertion procedure and ensuring that the nursing staff caring for the patient are trained in the use of the infusion device.

You are the SPR on an emergency list and are finishing a case on a 61-year-old man at 3 am. The original surgical plan was for a laparoscopic resection of a portion of large bowel, due to obstruction caused by a likely tumour. At the WHO team huddle, the surgeons discussed the possibility of converting to open and so you consented the patient and performed a spinal anaesthetic with intrathecal opioid prior to induction of general anaesthetic. You had also discussed the option of further regional techniques if required. The surgeons did need to convert to an open procedure, and the operation proceeded without further complication. The surgical team are now closing.

How would you manage this patient's postoperative analgesia?

This patient has had extensive surgery, with a likely significant pain burden. I would use a multimodal approach to analgesia, using the WHO pain ladder, adjuvants to analgesia

and regional techniques where possible. I would ensure intraoperative paracetamol and magnesium had been given.

Postoperatively I would ensure the patient receives regular paracetamol, consider NSAIDs (although there are likely contraindications in emergency surgery), and consider an IV morphine or fentanyl PCA. Other strategies to consider are clonidine, IV lidocaine or IV ketamine. Lidocaine or ketamine can be given as an intraoperative loading dose and run as a postoperative infusion; however, the patient should be cared for in an HDU setting while on such an infusion. For ketamine, care must be taken in patients at risk of postoperative delirium.

Regional options to supplement neuraxial intervention, include trunk blocks such as rectus sheath blocks, transversus abdominis plane (TAP) blocks or quadratus lumborum (QL) blocks. If the patient had had a midline laparotomy, I would choose a rectus sheath block, as this would also allow the opportunity for placement of catheters under the rectus sheath and continuous infusion of local anaesthetic for prolonged analgesia. The catheters can be placed either by the surgeons under direct vision, or postoperatively, prior to extubation, under ultrasound guidance.

How would you prepare to insert rectus sheath catheters?

Prior to the end of the operation I would let the ODP and the theatre team know about the intended procedure prior to extubation. I would discuss the plan with the surgical team and ensure no extra local anaesthetic is administered by them, to avoid exceeding the maximum dose. Then I would prepare the relevant equipment; an ultrasound machine, a sterile procedure pack, surgical gown, gloves, antiseptic skin preparation, a sterile probe cover and sterile gel, the catheters and a sterile insertion needle. I would check for any contraindication or allergies.

I would perform the procedure with the patient still anaesthetised and ventilated with full AoA monitoring, in the supine position. This is a sterile procedure, so I would be scrubbed and prepare a sterile field prior to insertion. I would use a high frequency linear transducer and initially set the depth to 4–5 cm. I would optimise the ergonomics prior to starting, placing the US machine on the other side of the patient's abdomen so I can clearly see it throughout.

Describe how you would insert rectus sheath catheters, including a description of the ultrasound anatomy.

I would place the ultrasound probe above the umbilicus in a transverse orientation, starting just off the midline. From superficial to deep I would expect to see: subcutaneous tissue, the anterior rectus sheath, the rectus abdominis muscle, the posterior rectus sheath, the peritoneum and the peritoneal cavity. To help me identify these structures, I could move my probe centrally to identify the linea alba, and the rectus abdomini muscles either side of it. The nerves that innervate the anterior abdominal wall run between the posterior rectus sheath and the rectus abdominis. I will aim to open this space with local anaesthetic and insert the catheters here.

Having identified the rectus abdominis and posterior rectus sheath, I would rotate my probe to a longitudinal orientation, and insert my needle in plane, pointing from a cephalad to caudal direction. When I can see the needle tip in the space between the rectus abdominis and the posterior sheath, I would hydro dissect the space by injecting

20 ml of 0.25% L-bupivacaine. Once I have opened the correct space, I would thread my catheter through the needle, aiming to leave 4–5 cm in the space. After repeating the procedure for the other side, I would safely secure and dress the catheters.

If you were unable to perform the rectus sheath catheters, can you describe the anatomy of different trunk block that you might consider?

I would think about doing a TAP block. The anterior abdominal wall is innervated by the anterior rami of spinal nerves of T6–T12, and the iliohypogastric and ilioinguinal nerves from L1. The aim of the TAP block is to deposit local anaesthetic in the transversus abdominis plane where these nerves travel before providing sensory innervation of the anterior abdominal wall.

I would place my ultrasound probe in the midaxillary line in a transverse orientation, around the level of the umbilicus. From superficial to deep on my ultrasound screen I would expect to see; subcutaneous tissue, external oblique muscle, internal oblique muscle and the transversus abdominis muscle. If I move the probe posteriorly, I should be able to see the quadratus lumborum muscle. The transversus abdominis plane runs between the internal oblique and the transversus abdominis.

I would keep an in-plane approach, with my needle moving from anterior to posterior, aiming for the most posterior aspect of the TAP. If my block is successful, I should see lifting of the internal oblique from the external oblique muscle/fascia.

What local anaesthetic would you use?

As a plane block the TAP block requires volume to open up the space and provide spread to the target nerves. I would use 20–30 ml of 0.25% L-bupivacaine on each side, provided this does not exceed the patient's maximum dose.

Regional Anaesthesia

4.2.1 Brachial Plexus, Interscalene and Axillary Blocks – Pascal J Boddy and Laura A Beard

What clinical approaches do you know to block the brachial plexus?

For a complete answer to this question a demonstration of an understanding of the anatomy of the brachial plexus is important. This could be aided by a diagram (see Figure 4.2.1.1.) The examiners may interrupt, in which case there are four well-recognised approaches you should mention:

1. Interscalene
2. Supraclavicular
3. Infraclavicular
4. Axillary.

The brachial plexus is formed from the anterior primary rami of C5 to T1 and provides all motor and sensory innervation to the upper limb. The nerves that make up the plexus lie in a sheath that extends from the tubercles of the transverse processes of the cervical vertebrae to the axilla; it is within this sheath that we would inject our local anaesthetic. As these five roots pass between scalenus anterior and medius, C5 and C6 join to form

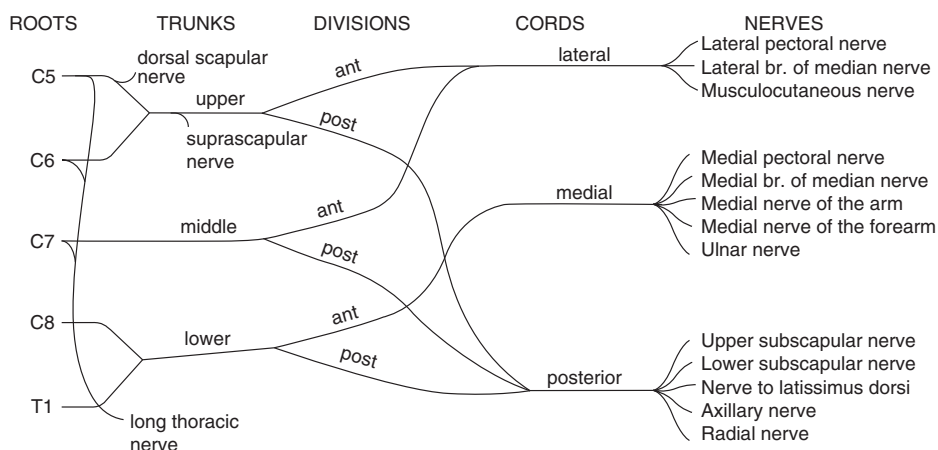


Figure 4.2.1.1 The brachial plexus.

the upper trunk, the root of C7 continues as the middle trunk and C8 and T1 unite to form the lower trunk. The interscalene approach aims to anaesthetise the roots of the plexus.

The three trunks continue downwards and laterally in the posterior triangle of the neck to cross over the first rib; the supraclavicular approach allows injection of local anaesthetic at this point.

At the lateral border of the rib the trunks each divide into anterior and posterior divisions (six divisions in total) and continue beneath the clavicle into the apex of the axilla to form the cords of the brachial plexus.

Within the axilla the lateral cord forms the lateral head of the median nerve and the musculocutaneous nerve. The medial cord forms the medial head of the median nerve and ulnar nerve. The posterior cord which is made up of the posterior divisions of all three trunks forms the radial nerve. The names of the cords correspond to their relationship to the axillary artery and is the level at which an infraclavicular brachial plexus block is performed. An axillary brachial plexus block provides the ability to block the plexus at the level of the branches, its most distal point.

For what type of surgery would an interscalene brachial plexus block be appropriate?

An interscalene block provides good analgesia for shoulder and upper arm surgery; however, ulnar sparing (C8 and T1) commonly occurs which makes this approach less suitable for hand surgery.

You have a patient listed for shoulder surgery, how would you perform an interscalene block?

I would firstly obtain informed consent from the patient and make sure the surgical site is marked and corresponds to the shoulder being operated on. I would ensure that full monitoring is established, that I have a trained assistant and that resuscitation equipment is easily available. I would then site an intravenous cannula in the contralateral arm.

The patient should be positioned supine, tilting their head slightly away from the side to be blocked. I would ensure asepsis by wearing a gown and sterile gloves and cleaning the skin with an appropriate cleaning solution. I would then undertake a prep, stop, block moment (previously called stop before your block) where the anaesthetic/surgical mark and patient consent are rechecked to ensure the correct side was selected.

A landmark or ultrasound-guided approach can then be undertaken. For a landmark approach, utilising a nerve stimulator, I would palpate for the interscalene groove at the level of the cricoid cartilage. If it was difficult to identify I could ask the patient to sniff which would make the scalene muscles appear more prominent. After anaesthetising the skin, I would pass a short-bevelled regional block needle attached to a nerve stimulator perpendicular to all planes. I may feel a 'pop' as the needle passes through the sheath at a depth of between 1 and 3 cm and should elicit muscle contractions in the biceps brachialis. At this point I would inject between 20 and 30 ml of local anaesthetic, ensuring that the injection was not intraneural by noting that there should be minimal resistance to injection and there should be minimal discomfort to the patient.

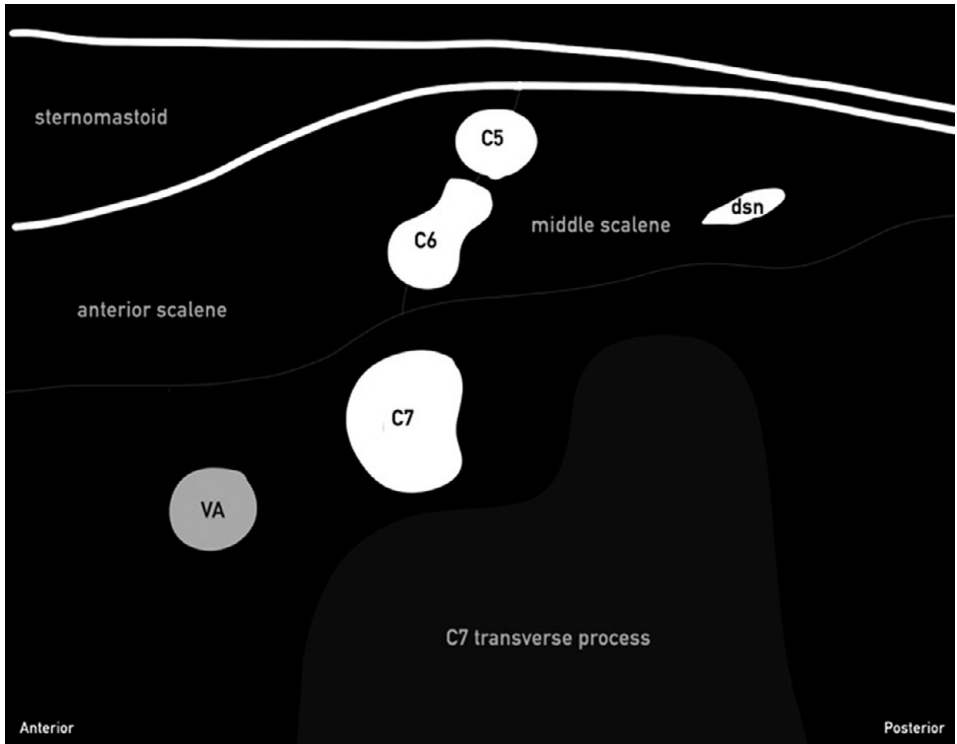


Figure 4.2.1.2 Schematic drawing of interscalene brachial plexus sonoanatomy.
Dsn – Dorsal scapula nerve, VA – vertebral artery

However, ultrasound-guided blocks are now commonly performed where the brachial plexus can be directly visualised. Using high frequency (10–13 Mhz) linear ultrasound probe I would identify the C5 and C6 nerve roots lying one on top of another between scalenus anterior and medius (see Figure 4.2.1.2). C6 commonly has a bifid appearance. Scanning up from the supraclavicular level can help with identification. I would use colour doppler to check for vascular structures such as the vertebral artery. The dorsal scapular nerves lies in the middle scalene and I would take care to identify and avoid this structure. The phrenic nerve can be seen on the surface on the anterior scalene muscle.

After anaesthetising the skin, using an in-plane approach with a 50 mm echogenic needle directed from the posterior end of the probe I would aim for the interscalene groove, being careful not to aim directly at the nerve roots. When injecting the local anaesthetic I would check there was no blood aspirated, minimal resistance to injection and that spread of local anaesthetic could be clearly seen on ultrasound. There should also be minimal patient discomfort if block performed awake. I would inject 15–20 ml of local anaesthetic. Volumes larger than this up to 30 ml can be used but increase the risk of phrenic nerve palsy. An out of plane approach is useful for catheter insertion.

You should try and make it sound that you are used to performing practical procedures and have a routine of following basic safety and infection control procedures. Mentioning consent and monitoring etc. also gives you some ‘thinking time’ before embarking on the

description of the procedure. The same preamble can be used for almost any practical clinical task.

What are the complications of an interscalene block?

Due to the level of the injection and the proximity of major cervical structures, complications can be very serious and life-threatening. They include:

- Cervical epidural or intrathecal injection
- Intravascular injection, particularly vertebral artery, carotid artery, external and internal jugular veins.

Less serious complications arise from the close proximity of other nerves and occur so frequently that they could be considered as effects of an interscalene block, and include:

- Phrenic nerve palsy (bilateral interscalene blocks should be avoided)
- Cervical sympathetic block (Horner's syndrome).

How would you reduce the risk of these complications?

Being aware of the depth of the plexus at this level, it is rarely deeper than 3 cm and is sometimes palpable in thin individuals.

Undertake a prep, stop, block moment before inserting the needle when the anaesthetic/surgical mark and consent are checked. This aims to reduce wrong site block which is a Never Event.

NICE recommendations suggest the use of ultrasound to aid needle placement in regional anaesthesia.

Lower volumes of local anaesthetic injectate reduce the risk of phrenic nerve palsy.

Could you employ another approach to anaesthetise the shoulder?

Other ultrasound-guided alternatives include

1. Superior trunk block
2. Axillary nerve block and Suprascapular nerve block
3. The supraclavicular approach can be used for shoulder surgery but as the suprascapular nerve (sensory to the shoulder joint) arises from the upper trunk, it may be missed especially if the nerve branches off earlier than normal. However, analgesia can be supplemented by a separate suprascapular nerve block.

How is a supraclavicular block performed?

Undertaking the same conditions as for interscalene block i.e patient consent, IV access, prep stop block, I would position the patient supine, arm adducted with their head turned away from the site of injection.

Utilising a landmark approach with nerve stimulator, the interscalene groove is palpated until the subclavian artery pulsation is felt. The injection is made just lateral and posterior to the subclavian artery. A pop should be felt as the needle pierces the sheath and nerve stimulation will lead to contraction of muscles in the forearm and hand. The volume of local anaesthetic required is usually between 20 and 30 ml. The first rib lies beneath the plexus at this point and serves a useful purpose in preventing the needle going too deep.

Ultrasound is useful to identify the plexus and direct the needle away from pleura and the subclavian artery and is why ultrasound is commonly used for this block. Using a high frequency (10–13Mhz) linear ultrasound probe I would place the probe above the clavicle in the supraclavicular fossa. I would then identify the pulsatile subclavian artery; colour doppler can be used. The brachial plexus can be seen posterior and superior to the artery; its appearance resembles a ‘bunch of grapes’. I would obtain the optimum image by rotating/tilting the probe until the first rib can be seen, the rib will help to protect underlying pleura and lung. The plexus is very superficial (1–4 cm). Using a 50mm needle with in-plane technique from lateral to medial I would deposit local anaesthetic around the plexus (10–30 ml), ensuring local anaesthetic is deposited in the lower part of the plexus near to the artery (the ‘corner pocket’) so that ulnar sparing does not occur.

What problems can arise from a supraclavicular block?

- Pneumothorax due to the proximity of the dome of the pleura the incidence can be as high as 1–2%, although by using ultrasound this is reduced dramatically.
- Perivascular injection is also a common complication, again reduced by the use of ultrasound. (The transverse cervical and dorsal scapular arteries may lie close to the plexus. Ultrasound colour doppler can help to identify these structures).
- Diaphragmatic paralysis can still occur due to the proximity of the phrenic nerve although the incidence is less than for interscalene blocks.
- Partial ulnar nerve block. The lower trunk can be difficult to approach using this technique.
- Suprascapular nerve can be missed and may need to be anaesthetised separately for shoulder surgery.

In what circumstances would you use a supraclavicular block?

Any upper limb surgery, as this technique provides good analgesia from the mid humerus to the hand as well as providing relief from tourniquet pain.

How is an axillary block performed?

The patient is positioned supine with their arm abducted to 90° and the elbow flexed. The patient’s hand is either held aloft or rests behind their head.

For a landmark/nerve stimulator approach the axillary artery is palpated and the needle is inserted just superior to the artery at the lateral border of pectoralis major. The response elicited with a nerve stimulator should be finger flexion (median nerve). Gentle distal pressure is applied to encourage medial spread of local anaesthetic and 30–40 ml is usually required. It is possible to identify and block the ulnar and occasionally the radial nerve in the axilla, but it can be time consuming, risks arterial injury and does not give a better result than a single large volume injection. It is, however, important to block the musculocutaneous nerve separately by injecting local anaesthetic into the coracobrachialis just superior to the axillary artery, or by infiltrating anaesthetic subcutaneously around the upper arm.

Frequently these blocks are now performed using ultrasound guidance. With the same patient positioning as above I would apply a high frequency linear probe transversely across the axilla. I would identify the pulsatile axillary artery, compressible

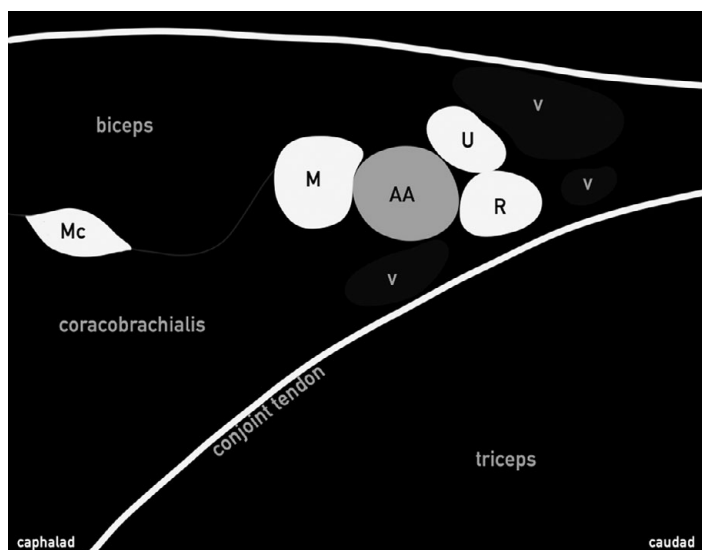


Figure 4.2.1.3 Schematic drawing of axillary brachial plexus sonoanatomy. Mc musculocutaneous nerve, M median nerve, U ulnar nerve, R radial nerve, V veins

axillary veins and hyperechoic conjoint tendon. The musculocutaneous nerve often lies intramuscularly between biceps and coracobrachialis (see Figure 4.2.1.3). The median, ulnar and radial nerves lie around the axillary artery at 11, 2 and 5 o'clock positions, however there is anatomical variability, and the nerves are not always in these positions so careful scanning is needed. I would use an in-plane technique with a 50–80 mm needle and between 20–30 ml of local anaesthetic.

In what circumstances is an axillary block useful?

An axillary block is relatively easy to perform and is useful for providing analgesia in patients with forearm and hand injuries. Compared to the other approaches to the brachial plexus there is no danger of causing a pneumothorax or phrenic nerve palsy. Tourniquet pain can be an issue as the intercostobrachial nerve which supplies the skin to the upper medial and posterior aspect of the arm is not blocked. The nerve is difficult to visualise with ultrasound due to its small size. Subcutaneous infiltration of local anaesthesia in the axilla can block this nerve.

What do you know about the infraclavicular approach to the brachial plexus?

If the structured oral exam is going well you could be asked about topics that you are less familiar with and may never have seen. Having even a basic knowledge of an unfamiliar and less common procedure is likely to impress the examiners.

The infraclavicular approach is less commonly performed in the UK, but is a popular approach on the continent and in America; it is also useful for the insertion of catheters due to its depth and surrounding musculature that help to hold catheters in place.

A linear probe can be used but a curved array probe may be more useful as the cords of the brachial plexus are often visualised at 4–6 cm depth. The probe is placed below the clavicle medial to the coracoid process. The three cords (medial, lateral and posterior) can be seen around the axillary artery. An 80 mm needle is advanced in plane under the clavicle. A single injection technique can be successful if local anaesthetic spread is seen below and around the artery.

This block provides excellent analgesia for elbow, forearm and hand surgery, but is unsuitable for shoulder and upper arm analgesia. Its deeper location can make identification of structures and needle tip visualisation challenging.

What current would you accept on your nerve stimulator when identifying the brachial plexus?

For landmark techniques using a nerve stimulator, a response with a current of 0.3–0.4 mA suggests that the tip of the stimulator needle is in close proximity to the target. A higher current would indicate that the target is still some distance from the needle tip and may not be anaesthetised after local anaesthetic injection. A higher current can also produce required responses with the needle still being outside the plexus sheath. If a response is elicited at a current less than 0.2 mA there is a danger that the needle tip is within the nerve, injection at this point could lead to permanent nerve damage. It is important to realise that nerve stimulators cause muscles to contract if directly stimulated which can lead to misinterpretation of the response observed.

Nerve stimulators can be used in combination with ultrasound. They are often set at a current of 0.4 mA and used as an additional safety method rather than for seeking nerve responses as for landmark techniques.

After performing a brachial plexus block, what can be done if the block is inadequate?

You should assess whether the block has failed completely or if there is an incomplete block. Complete failure should lead the operator to try an alternative type of anaesthesia; it is inadvisable to attempt the same block again as the anatomy may have been transiently disrupted making further attempts less likely to succeed. The dose of local anaesthetic already injected should be considered, as a repeat injection may lead to an excessive dose of local anaesthetic being given. Forearm and wrist blocks of the nerves not anaesthetised can supplement an incomplete block.

What are the contraindications to brachial plexus blocks?

Absolute contraindications include:

- Patient refusal
- Allergy to local anaesthetic
- Infection at the site of injection
- Severe coagulation disorders.

Relative contraindications include:

- Severe respiratory compromise. The high incidence of phrenic nerve palsy with root blocks such as interscalene and supraclavicular blocks could result in decompensation and lead to respiratory failure.

- Mild to moderate coagulation disorders. The choice of approach may be governed by its proximity to major blood vessels, and how easy it would be to apply pressure to a bleeding point.

Presence of anticipated technical difficulties such as anatomical variation:

- Lack of equipment.
- Inexperienced operators - Regional Anaesthesia UK (RAUK) have recently endorsed a group of Plan A blocks that a general anaesthetist could focus on perfecting. For upper limb this includes interscalene and axillary plexus blocks.

Further Reading

Al Haddad MF, Coventry DM. Brachial plexus blockade. *British Journal of Anaesthesia*. CEPD Reviews 2002; 2(2): 33–36.

Carty S, Nicholls B. Ultrasound-guided regional anaesthesia. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2007; 7(1): 20–24.

Hewson DW, Oldman M, Bedforth NM. Regional anaesthesia for shoulder surgery. *BJA Education*. 2019; 19(4): 98–104.

Macfarlane A, Anderson K. Infra-clavicular brachial plexus blocks. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2009; 9(5): 139–143.

Pavan Kumar BC, Coventry DM. Ultrasound-guided brachial plexus blocks. *British Journal of Anaesthesia*. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2014; 14: 185–191.

Regional Anaesthesia UK (RAUK) www.ra-uk.org (Plan A Blocks).

Turbitt LR, Mariano ER, El-boghdadly K. Future directions in regional anaesthesia: Not just for the cognoscenti. *Anaesthesia*. 2020; 75(3): 293–297.

Wingate R, Foxall G, Russon K. Ultrasound guided axillary brachial plexus block. *Anaesthesia Tutorial of the Week*. 2016; (ATOTW 326).

4.2.2 Stellate Ganglion and Coeliac Plexus Blocks – Rosie Trumper and Jonathan J Gatward

Can you describe the anatomy of the stellate ganglion?

Before the SOE, learn to draw a simple line diagram to describe the anatomy. This will help you to remember the position and relations of the ganglion, and can be quickly reproduced in the exam, rather than trying to recall the information verbally (see Figure 4.2.2.1).

The stellate (or cervicothoracic) ganglion consists of a fusion of the inferior cervical and first thoracic ganglia of the sympathetic chain. It lies adjacent to the vertebral column, between the carotid sheath and the fascia overlying the prevertebral muscles at the level of the seventh cervical and first thoracic vertebrae. It is closely related to the neck of the first rib, and importantly, the dome of the pleura and the vertebral artery which both lie anterior to it. It is important to note that the stellate ganglion is absent in up to 20% of the population.

Can you describe a technique for performing a stellate ganglion block?

You may not have performed a stellate ganglion block. You will find it easier to remember the surface anatomy if you have at least attempted to palpate Chassaignac's tubercle. Try it out! When describing the ultrasound-guided procedure, a simple diagram may be useful (see Figure 4.2.2.2).

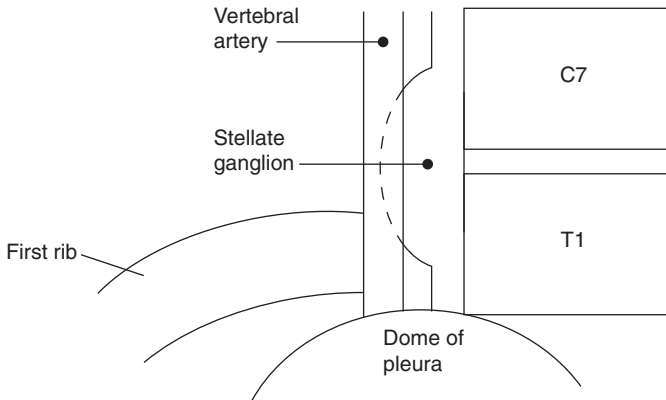


Figure 4.2.2.1 The stellate ganglion.

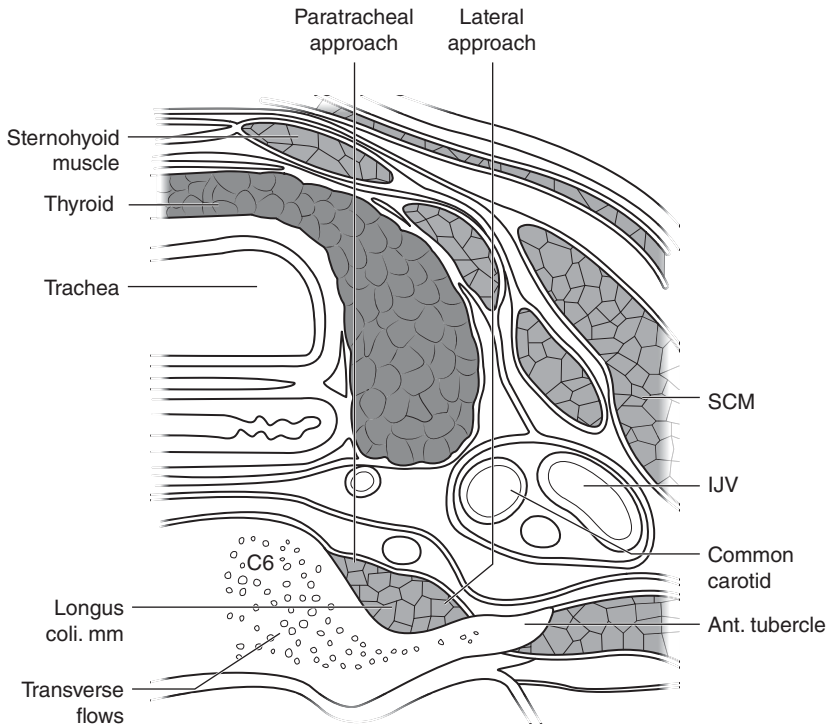


Figure 4.2.2.2 Anatomy and needle path for the ultrasound-guided stellate ganglion block.

Blockade of the stellate ganglion can be performed using a landmark technique with fluoroscopy, or as an ultrasound-guided procedure. The use of ultrasound guidance has become the preferred method over the past decade, owing to the increased success rate and reduction in risk of complications such as inadvertent intravascular injection,

pneumothorax and direct needle trauma to other surrounding structures. The aim is to place local anaesthetic solution at C6 with caudal spread blocking the ganglion, because a direct approach risks entering the dome of the lung or the vertebral artery.

First, I will describe the anterior approach to the ganglion using a landmark technique. The patient is placed in the supine position, with the neck extended and rotated away from the side of the block. The carotid pulsation is palpated at the level of the cricoid cartilage. The sternocleidomastoid muscle and the carotid artery are displaced laterally so that the transverse process of C6 can be palpated. This is known as Chassaignac's tubercle.

After injecting local anaesthetic into the skin, a 25-gauge needle is inserted at 90 degrees to the skin and advanced towards the tubercle until bone is contacted. It is then withdrawn by 2 mm. Position should be confirmed fluoroscopically, in which case a contrast dye may be injected. It is very important to aspirate for blood before injecting 0.5 ml of solution as a test dose. An adrenaline containing test dose may be used to rule out intravenous placement. If there are no adverse effects, 15 to 20 ml of low concentration local anaesthetic solution, such as 0.5% lidocaine or 0.125% bupivacaine is injected in 5 ml increments. It is best to sit the patient up to aid spread down towards the stellate ganglion.

With ultrasound-guided blockade of the stellate ganglion, positioning of the patient remains the same but a lateral approach is taken to reach the site of injection. The linear ultrasound probe is placed on the antero-lateral aspect of the neck at the level of C6, on the side to be blocked. The needle is then inserted lateral to the probe and advanced medially, in plane, towards the anterior surface of the C6 transverse process. The local anaesthetic is infiltrated, under vision, just superficial to the longus colli muscle. When positioned correctly, injection of fluid will lead to separation of the prevertebral fascia from the carotid sheath. A total volume of 10–20 ml is then injected in small aliquots.

Signs of a good block are the same as the signs of Horner's syndrome: ipsilateral ptosis, miosis, anhydrosis, conjunctival injection and enophthalmos. Patients may also get a blocked nose on the same side due to vasodilatation of the nasal mucosa (known as Guttman's sign). Warm, dry skin on the arm and hand suggests a good sympathetic block to the upper limb.

Can you name some indications for a stellate ganglion block?

Remember to categorise your answer.

Firstly, the stellate ganglion can be blocked to treat neuropathic pain, for example:

- Complex regional pain syndromes (CRPS) of the upper limb
- Acute herpes zoster and post-herpetic neuralgia
- Central pain following stroke
- Phantom limb pain.

The stellate ganglion block can also be used to treat ischaemic conditions in the upper limb such as:

- Vascular insufficiency
- Raynaud's disease
- After vascular or re-implantation surgery
- Inadvertent intra-arterial injection of thiopentone.

Other indications include:

- Treatment of visceral pain from the heart in refractory angina.
- Temporary treatment of refractory VT or VT storm while awaiting sympathectomy
- Hot flushes, hyperhidrosis and PTSD (however, there is limited evidence for this).

What are the contraindications to stellate ganglion blockade?

General contraindications applicable to any regional block include patient refusal, allergy, overlying infection, anticoagulation or coagulopathy.

Contraindications specific to stellate ganglion block include contralateral recurrent laryngeal or phrenic nerve palsy, contralateral respiratory compromise (for example, severe COPD or pneumothorax) and glaucoma.

Why are sympathetic blocks thought to work in the treatment of neuropathic pain?

The sympathetic nervous system seems to have a role in the generation of pain. In certain pain states, there is an abnormal response of the primary nociceptive afferents to sympathetic stimulation. Peripheral and central sensitisation occurs, with reduced excitatory thresholds and the production of ectopic impulses. The afferent fibres also have increased adrenoceptor sensitivity, so that noradrenaline released from sympathetic nerve terminals causes further sensitisation. This sympathetically mediated pain is sometimes seen in CRPS. Sympathetic blocks are often used in CRPS for that reason, though only about a third of patients with the condition have sympathetically mediated pain.

Can you describe the specific side effects and complications of the stellate ganglion block?

The side effects of a stellate ganglion block include blocking other nearby nerves, such as the recurrent laryngeal nerve causing hoarseness, the vagus nerve causing difficulty swallowing, branches of the cervical or brachial plexus causing motor and sensory deficits, and phrenic nerve palsy, which is why bilateral blocks should not be performed at the same time.

Inadvertent injection can also occur, for example, into the vertebral or carotid arteries causing seizures, and intrathecal injection causing a spinal block.

Other complications include damage to blood vessels causing haematoma, damage to the vagus nerve or brachial plexus, the dome of the pleura causing pneumothorax, oesophageal puncture and chylothorax. Infection can also be introduced, causing abscess, osteitis or meningitis.

These complications can be minimised using real-time ultrasound guidance with a lateral approach at the level of C6.

Let's move on to a different part of the body now. Can you describe the anatomy of the coeliac plexus?

Again, you may never have performed a coeliac plexus block but you need to know the anatomical relations of the plexus. Practise drawing a simple cross-sectional diagram of the abdomen at the level of the first lumbar vertebra (see Figure 4.2.2.3).

The coeliac (or solar) plexus is the main junction for autonomic nerves supplying the upper abdominal organs and consists of the bilateral coeliac ganglia with a network of

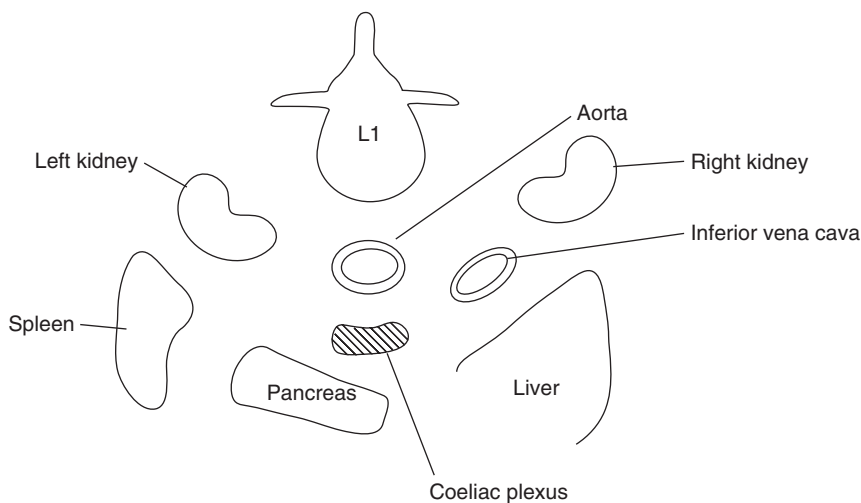


Figure 4.2.2.3 The coeliac plexus.

interconnecting fibres. Some sympathetic preganglionic fibres do not synapse within the sympathetic chain, but exit the chain as a splanchnic nerve and synapse in prevertebral coeliac ganglia. The coeliac plexus is made up of fibres from T5 to T10, which form the greater splanchnic nerve, fibres from T9 to T11 which form the lesser splanchnic nerve, and fibres from T12 which form the least splanchnic nerve. The plexus also receives fibres from the vagus nerve. The coeliac ganglia lie anterior to the aorta on either side of the body of L1, and posterior to the pancreas.

Why might we want to perform a coeliac plexus block?

The sympathetic plexuses receive nociceptive impulses from all viscera via unmyelinated afferent fibres. These impulses can be blocked with local anaesthetic or neurolytic techniques. The coeliac plexus receives afferents from the pancreas, so the block is usually performed for pain associated with pancreatic cancer or pancreatitis. There is a risk of paraplegia of about 2 per 1000 blocks, so the risks and benefits must be balanced carefully. Also, the block may only last for a few months, and so may need to be repeated.

Do you know what a diagnostic block is?

A diagnostic block is performed with local anaesthetic only and is used to try to locate the source of pain or investigate how amenable it will be to treatment. If it is successful, there is the possibility of progressing to therapeutic or neurolytic treatment in the future.

What agents can we use in a therapeutic block?

We can use local anaesthetic agents with or without corticosteroids.

Why do therapeutic blocks with local anaesthetic alone last longer than the expected duration of action of local anaesthetic agent?

This may be due to the prevention of reflex muscle or sympathetic nervous effects, breaking the cycle of sympathetically mediated pain. Also, they may cause reduced

sprouting of neurones in spinal ganglia, reduced ephaptic transmission (electrical transmission across the synapse which is not neurotransmitter mediated), and reduced hyperexcitability of pain fibres.

How can we achieve a neurolytic block?

We can use chemical, thermal or cryogenic techniques to destroy nerve tissue. The commonly used chemical agents are phenol 5 or 6% or ethanol 50 to 100%. These chemicals must be used with care, as they are obviously toxic to all tissue, not just the nerves we are targeting. Ethanol causes severe pain on injection, so it is important to perform an adequate therapeutic block prior to neurolysis with this agent.

How would you prepare a patient for a coeliac plexus block?

Firstly, I would take a medical and anaesthetic history and perform a clinical examination. I would gain informed consent and discuss the risks and benefits of the treatment, including the potential side effects and complications. I would ensure that there are no contraindications, such as local infection or coagulopathy. I would secure intravenous access, fluid load the patient to reduce the risk of hypotension and make sure that full airway and resuscitation equipment was immediately available. I would attach the patient to standard monitoring and ensure that I had a skilled assistant. The block should be done under strict aseptic conditions, using antiseptic solution, sterile gloves, mask and gown, and sterile, single-use needles. The block also requires the use of an image intensifier with contrast (and/or ultrasound if performing an anterior percutaneous approach) to confirm correct needle placement.

Your patient is prepared and you are scrubbed, with a radiographer and image intensifier in position. Can you describe the technique for performing a coeliac plexus block?

There are several different methods for performing a coeliac plexus block, involving various approaches. These include retrocrural and transcrural approaches, as well as the anterior percutaneous approach which involves needle passage through the abdominal wall and liver. Coeliac plexus block can also be performed via an endoscopic ultrasound-guided approach.

I will describe the classic retrocrural approach. The patient is in the prone position. The insertion point is just below the tip of the twelfth rib, approximately 8 cm lateral from the midline. The block is bilateral. After local anaesthetic infiltration of the superficial layers, a 100 to 150 mm needle is inserted and directed medially towards the body of L1. The needle is inserted until it comes into contact with bone (the side of the body of L1) and then it is withdrawn and redirected anteriorly. It is then advanced a further 2 to 3 cm. Position is confirmed with radio-opaque dye spreading caudad and cephalad in the paracolic gutter, with no lateral spread. It is really important to aspirate before injecting. After a small test dose, 10 ml of local anaesthetic solution, with or without a neurolytic agent is then injected, under image intensifier guidance on each side.

What are the specific complications associated with the coeliac plexus block?

Remember to categorise your answers.

The complications can be divided into those associated with a misplaced needle and those caused by the drugs used.

The needle can enter the inferior vena cava, the aorta or the coeliac artery, causing retroperitoneal haemorrhage. The kidneys or adrenal glands can be damaged, as can any upper abdominal organ, with abscess or cyst formation.

Complications caused by the drugs used include hypotension due to sympathetic blockade, even after unilateral block. Paraplegia can occur from injecting phenol into the arteries that supply the spinal cord. Sexual dysfunction is an important complication, which can occur if injected solution spreads to the sympathetic chain bilaterally. Finally, lumbar nerve root irritation can occur if injected solution tracks back towards the lumbar plexus.

4.2.3 Ulnar, Median and Radial Nerve Blocks – Poonam M Bopanna and Laura A Beard

The nerves of the arm can be blocked at a number of distal sites. You should have good knowledge of the anatomy and approaches for all the common peripheral nerve blocks. There are some considerations relevant to all blocks, so it is useful to learn a list and mention the points if you are asked about any block.

Points to consider when performing nerve blocks:

- Relevant medical and anaesthetic history including any drug allergies, airway assessment and aspiration risk
- Patient (and surgical) consent to the block
- Contraindications
 - History of infection at the site of injection
 - Anticoagulation (may be relative or absolute contraindication)
 - Patient refusal
 - Allergy
- IV access required in the contralateral limb
- Full airway and resuscitation equipment must be available (AoA recommended standard monitoring)
- Trained assistant present
- Undertake prep, stop, block check before performing block (previously known as stop before you block).

How would you perform ultrasound-guided nerve blocks of the upper limb?

Ultrasound-guided peripheral nerve blocks can be used to provide anaesthesia for hand surgery, as 'rescue' blocks if there has been sparing from a proximal brachial plexus block or for postoperative analgesia. Clear knowledge of distal limb innervation and the surgical procedure being performed is needed.

Typically 3–5 ml of local anaesthetic is needed per nerve to achieve sensory block. Due to their superficial location a 50mm needle and high frequency linear probe are used.

Radial Nerve:

The patient is positioned supine, with arm adducted, elbow flexed and hand resting on the abdomen. The probe is placed transversely over the lower third of the humerus. If you scan along the humerus from proximal to distal the radial nerve can be seen moving from posterior to anterior in the spiral groove. The nerve divides into superficial and deep branches at the level of the elbow. Blockage of the deep branch is needed for bony surgery at the wrist.

Below the elbow with the arm extended and supinated the radial nerve can be located on the lateral side of the radial artery; however, blocking this distally would provide only cutaneous anaesthesia.

Median Nerve:

With the arm extended and supinated the median nerve lies medial to the brachial artery near to the antecubital fossa. The needle is directed in-plane from the medial side of the arm to avoid the brachial artery. More distally it can be identified between flexor digitorum superficialis and flexor digitorum profundus.

Ulnar Nerve:

Position the patient with arm abducted and elbow flexed. The ulnar nerve can be identified on the medial side of the distal humerus above the medial epicondyle. Do not block the nerve as it enters the cubital tunnel as this can result in nerve injury from inadequate room for tissue expansion.

Distally in the forearm the ulnar nerve can be identified medial to the ulnar artery.

Musculocutaneous nerve:

With the arm abducted the musculocutaneous nerve can be identified and blocked in the axilla in the plane between biceps and coracobrachialis muscles as for an axillary brachial plexus block (see Section 4.2.1).

Further Reading

Capek A, Dolan J. Ultrasound guided peripheral nerve blocks of the upper limb. *BJA Education*. 2015; 15(3): 160–165.

Snaith R, Dolan J. Ultrasound-guided peripheral upper limb nerve blocks for day case surgery. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2011; 11(5): 172–176.

4.2.4 Caudal Block – Corinna J Hughes and Philip Harrington

This topic may come up as part of a short case, a discussion about anatomy or even as part of a long case when discussing operative planning.

Can you tell me about caudal analgesia?

Caudal analgesia is produced by injection of local anaesthetic into the caudal canal to block lumbar and sacral nerve roots. It is mainly used in paediatric anaesthesia where it

represents the most common form of regional anaesthesia in children. It is popular due to its low failure rate, complication rate and favourable side effect profile.

Can you tell me about the anatomy?

Draw a sagittal section diagram (Figure 4.2.4).

The caudal space is the lowest part of the epidural space contained by the sacrum.

The sacrum consists of five fused sacral vertebra.

It articulates with:

- 5th lumbar vertebra superiorly
- The coccyx inferiorly
- The ilia laterally.

The dorsal roof has a median crest in the midline from fusion of the sacral spinous processes. Failure of fusion of S5 (and occasionally S4) leaves a defect called the sacral hiatus. The sacrococcygeal membrane covers this. On either side of the sacral hiatus are sacral cornua. The sacral canal is accessed via the sacral hiatus.

What does the sacral canal contain?

The contents of the sacral canal are:

- The terminal part of the dural sac, which finishes at approximately S1–2 in adults and S3–4 in infants

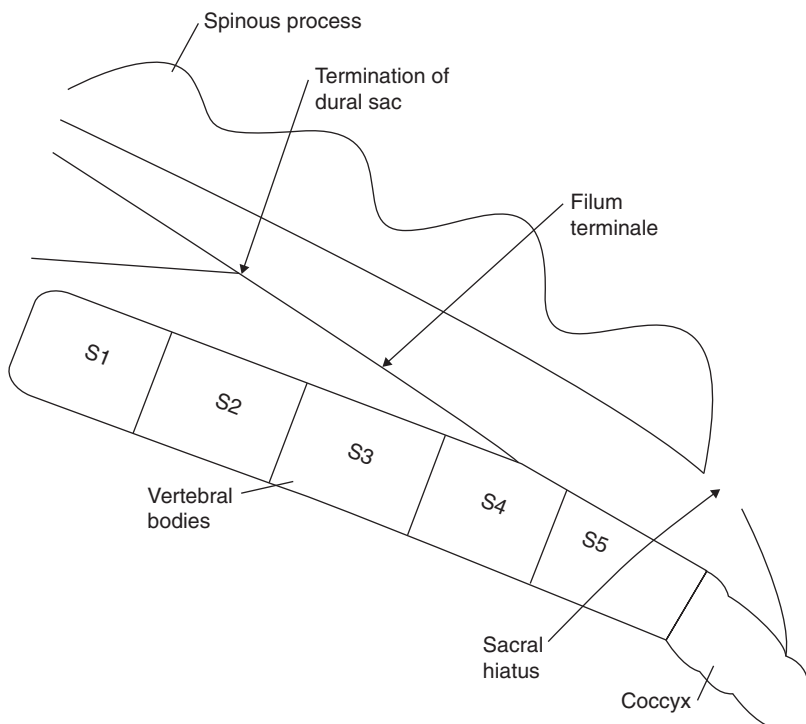


Figure 4.2.4 The caudal epidural space.

- The cauda equina, which consists of five sacral and coccygeal nerves
- The filum terminale
- The venous plexus
- Epidural fat.

What operations can caudal analgesia be used for?

In adults, caudal blocks are mainly used for operations on the perineum. In children they are used more extensively for operations below the umbilicus. These include inguinal hernia repair, hypospadias repair, orchidopexy, and lower limb surgery.

Why are they used more extensively in children than in adults?

The fat within the sacral canal is loose in children allowing the spread of local anaesthetic making the block much more predictable. In adults the connective tissue forms a fibrous, closed mesh that leads to an unpredictable spread of local anaesthetic. The volume of the sacral canal also varies greatly in adults. The sacral cornua are more easily identifiable in children.

Can you describe your technique?

The examiners will probably move you on but don't forget to start by mentioning preparations such as preoperative assessment, consent and all safety precautions. Make sure you have done some caudal blocks so you can speak convincingly about your own technique.

I would preoperatively assess the patient, gain consent, obtain IV access and full monitoring, ensure I had a trained assistant and emergency drugs and equipment. I follow strict aseptic procedure. In children I perform the block after induction of anaesthesia.

The steps I follow to carry out the block are:

- Position the patient is left laterally with knees flexed.
- Identify the landmarks by finding the posterior superior iliac spines and putting the left thumb and index finger on them. The sacral hiatus forms the third point of an equilateral triangle. It may not lie in the natal cleft. The sacral cornua can then be palpated by the left index finger.
- Use a 20 or 22-gauge cannula and insert the cannula at 45 degrees cranially, while feeling the sacral cornua.
- Proceed until a click is felt as the cannula goes through the sacrococcygeal membrane.
- Advance the cannula over the needle.
- Remove the needle.
- Exclude the presence of blood or cerebrospinal fluid by gently aspirating (not too hard as this may yield a negative aspiration due to collapse of the vein if the cannulae is intravenous).
- Slowly inject the local anaesthetic, ensuring there is no resistance, no subcutaneous swelling and intermittently aspirating.

What dose of local anaesthetic do you use?

In adults, 20–30 ml of bupivacaine 0.25–0.5% depending on the size of the patient. The volume of the sacral canal is normally 30–35 ml.

For children the volume depends on the level of block required and is classically dosed using the Armitage regimen.

For a sacral block	0.5 ml/kg 0.25% bupivacaine
For lumbar block	1 ml/kg 0.25% bupivacaine
For thoracolumbar block	1.25 ml/kg 0.19% bupivacaine

How do you get 0.19% bupivacaine?

Don't give information about things that YOU do without being able to back it up.

1 part normal saline to 3 parts 0.25% bupivacaine.

How long do caudal blocks generally last?

There is a lot of individual variety, if you don't know then guess! Giving information about additives looks like you've thought about how to prolong your block.

They normally last 4–8 hours. They can be extended by using a catheter technique or by using additives. These include:

- Clonidine (1 microg/kg)
- Opioids such as diamorphine at 30 microg/kg
- Preservative free ketamine at 0.5 mg/kg.

Are there any problems with these additives?

We must ensure that they are preservative free, as preservatives may be neurotoxic. Clonidine and opioids can cause sedation. Opioids may cause respiratory depression and urinary retention. I do not use additives for daycase procedures.

What complications are there?

Give figures if you can.

Severe complications are rare using a single-shot technique.

- Failure
- Urinary retention (4–8%)
- Leg weakness (4–8%)
- Proprioception loss (4–8%)
- Inadvertent intravenous (1:10 000)
- Inadvertent intradural injection
- Epidural abscess or haematoma

The widespread development of regional anaesthesia complemented by the use of ultrasound has meant a gradual move away from central neuraxial blockade. Often there is a peripheral nerve that can be blocked which avoids some of the complications listed above. However, on a case-by-case basis there still may remain good reason for performing a centrally acting block and this will need consideration of both the patient and surgical procedure being performed.

Further Reading

Greaney D, Everett T. Paediatric regional anaesthesia: Updates in central neuraxial

techniques and thoracic and abdominal blocks. *British Journal of Anaesthesia*. 2019; 19(4): 126–134.

4.2.5 Ankle and Knee Blocks – Matthew P Morgan and Andrew Weir

This topic would be well suited to the anatomy section of the clinical sciences SOE.

What nerves supply the structures below the knee?

The nerve supply to structures below the knee is predominantly derived from the sciatic nerve, although the saphenous nerve supplies a variable portion of the medial calf. The sciatic nerve, derived from L4 to S3, divides approximately 6 cm above the popliteal skin crease into the tibial and common peroneal nerves. The tibial nerve subsequently divides into the medial and lateral plantar nerves beyond the medial malleolus and supplies the plantar surface of the foot. After passing around the neck of the fibula, the common peroneal nerve divides into the deep and superficial peroneal nerves. The superficial peroneal nerve supplies the majority of sensation on the dorsum of the foot, while the deep branch supplies a small area of skin webbing between the first and second toes.

Motor innervation of the ankle is derived from the common peroneal nerve, which causes dorsiflexion and eversion, while the tibial nerve results in plantar flexion and inversion. Finally, the sural nerve is formed from fibres of both the common peroneal and tibial nerves and supplies sensation of the lateral foot and calf.

What methods of nerve identification can be used in knee and ankle blocks?

In modern anaesthetic practice, identification of nerves for knee and ankle blocks relies predominantly on knowledge of the relevant anatomy and the use of ultrasonography, although ankle blocks are still often performed using a landmark technique. At the knee, a nerve stimulator can be combined with ultrasound to aid identification, although many anaesthetists will use ultrasound alone.

How do nerve stimulators used for the identification of peripheral nerves differ from those used for assessing neuromuscular blockade and why?

Due to the wide variation of tissue and hence impedance encountered as a nerve is approached by a stimulating needle, devices used for identification of peripheral nerves must be constant current generators. In addition, the delivered output should be clearly indicated on a digital display. The current duration, termed chronaxies, should be shortened to around 100 msec in order to preferentially stimulate A α motor fibres and not A δ pain fibres. These devices use an insulated needle to maximise and pinpoint delivered current while minimising peripheral current loss. Incorrectly attaching the positive electrode to the needle will result in a cone of hyperpolarisation around the nerve and hence reduce effectiveness.

How would you perform a popliteal nerve block and what are the causes of sub-optimal intraoperative analgesia?

While there are landmark techniques described to block the nerves of the popliteal fossa, I am most comfortable with an ultrasound-guided popliteal fossa block, which can be

performed in an awake or anaesthetised patient. After confirming that all relevant equipment and drugs were available, intravenous access was gained, and appropriate monitoring was applied, I would ensure a 'Stop Before You Block' technique was used to reduce the risk of wrong-sided block. With the patient supine and the knee bent to 90 degrees, I would start with a high frequency linear probe in the popliteal fossa and move superiorly until I visualised the division of the sciatic nerve into the tibial and common peroneal nerves, superficial to the popliteal artery and vein. At this point, using an in-plane approach with a short-bevelled ultrasound needle I would inject local anaesthetic around the nerves, such as 20 ml of 0.5% bupivacaine or similar, taking care not to inject intraneurally and I would visually ensure local anaesthetic spread around the nerves. If there were any doubt as to position, I would use a nerve stimulator to confirm the location of the nerves.

Sub-optimal analgesia can result from a poorly working block or a poorly selected block. A nerve block may fail for a number of reasons:

- Local anaesthetic may have been deposited in the wrong location. Intravascular injection would result in a poor block.
- The nerve block selected may not cover an adequate area for the surgery. For example, surgery on the medial surface of the foot may additionally require a saphenous nerve block to ensure adequate analgesia.
- Using an above knee tourniquet would require additional analgesia or a more proximal nerve block.

You perform a popliteal block using a nerve stimulator on a patient with long-term diabetes. One week later you are told that she has paraesthesia over the outer aspect of her upper thigh. What are the potential causes?

The causes of paraesthesia can be divided into those related to anaesthesia, those related to surgery and other causes. Let's start by considering those related to anaesthesia. The popliteal block will often be blamed for such problems; however, the pattern of injury makes this very unlikely. The distribution of paraesthesia suggests involvement of the lateral cutaneous nerve of the thigh at a point above where the previous block was performed. Damage to the lateral cutaneous nerve of the thigh can often be traced to poor intraoperative positioning, especially in people with conditions increasing chances of neuropraxias such as diabetes. Poor positioning results in neuropraxias due to a combination of direct nerve compression, ischaemia and nerve stretching.

Surgical causes would include direct nerve trauma although in this case tourniquet damage would be more likely. Finally, the paraesthesia may be unrelated to recent events and represent a mononeuropathy from diabetes alone.

What are the advantages of a peripheral nerve block over those placed more centrally?

There are advantages to the technique itself and advantages to the patient. In general, the more peripheral a nerve is blocked the more superficial it can be found and therefore landmarks become simpler. Additionally, superficial nerves are often more amenable to ultrasound imaging and possibly easier to identify with a nerve stimulator.

Turning to advantages for the patient, central nerve blocks tend to be placed closer to vascular and neurological structures, and thus they carry greater risks. In addition,

blocking nerves more peripherally will enable one to avoid some of the central effects of nerve blockade including urinary retention and dense whole limb weakness. For knee surgery, more distal blocks are often preferred compared to proximal femoral nerve blocks as they are 'motor sparing', and therefore allow mobilisation earlier after surgery.

Should a popliteal nerve block be performed in a patient with a distal tibia fracture?

There are advantages and disadvantages to using peripheral nerve blocks. A traumatic tibial fracture is known to carry a risk of compartment syndrome, which persists even after surgical fixation. Some may argue that performing a long-lasting peripheral nerve block would make identifying a compartment syndrome difficult. There have, however, been studies showing that ischaemia type pain, common in compartment syndrome, will break through even a perfectly working peripheral nerve block. It has also been shown that good postoperative analgesia and hence early mobilisation can reduce the incidence of compartment syndrome. It could be argued that if surgeons are sufficiently concerned about the risks of compartment syndrome to advise avoiding a peripheral nerve block, then a different surgical technique or postoperative compartment pressure monitoring should be used. A 2021 Association of Anaesthetists consensus statement recommended that dense blocks of long duration should be avoided, but that continuous or single-shot blocks with lower concentrations of local anaesthetic are not associated with delays in diagnosis. Overall, the decision to use a peripheral nerve block in these circumstances should be made jointly between the surgeon, anaesthetist and patient, and would be influenced by the safety of administering systemic analgesia to the patient in question.

When would you consider performing an ankle block?

I would consider an ankle block to provide surgical anaesthesia and postoperative pain relief for forefoot and digit surgery.

Which nerves are blocked in an ankle block?

There are five nerves that are blocked when performing an ankle block. Four of these nerves are terminal branches of the sciatic nerve:

- Tibial nerve
- Superficial peroneal nerve
- Deep peroneal nerve
- Sural nerve.

The remaining nerve, the saphenous nerve, is a branch of the femoral nerve.

Please tell me how you would block the superficial and deep peroneal nerves.

While there are ultrasound-guided techniques for ankle blocks, I have most commonly seen a landmark technique used. I would position the foot so that it was at right angles with the tibia. I would then identify the tendon of the extensor hallucis longus by moving

the big toe. The dorsalis pedis pulse is found lateral to the tendon. I would insert a 23- to 25-gauge needle 2–3 cm from the inter-malleolar line first medial and then lateral to the artery. I would advance the needle until contact is made with bone, I would then withdraw slightly and inject 2 ml on either side of the artery.

To perform a superficial peroneal nerve block I would withdraw the needle from the position it was in to perform a deep peroneal nerve block into the subcutaneous tissue. I would then infiltrate up to 10 ml of local anaesthetic laterally and medially across the dorsum of the foot to produce a weal of local anaesthetic to block the medial and lateral divisions of the superficial peroneal nerve.

How would you block the tibial nerve?

I would start by drawing a line between the medial malleolus and the posterior inferior border of the calcaneum. I would then palpate the posterior tibial pulse and mark a point on the line just posterior to the pulse. I would insert a 23-gauge, 50mm needle at 90 degrees to the skin until either plantar flexion of the toes is elicited by a nerve stimulator, or paraesthesia of the sole of the foot is experienced. A range of 6–10 ml of local anaesthesia is then injected.

How would you block the sural nerve?

The sural nerve is blocked by infiltrating 5 ml of local anaesthetic subcutaneously from the lateral malleolus to the lateral border of the Achilles tendon. A 23-gauge, 50mm needle should be used.

Finally, how would you block the saphenous nerve?

I would identify the saphenous vein anterior and proximal to the medial malleolus at the ankle. Using a 23-gauge needle, I would infiltrate 2 ml of local anaesthetic on either side of the saphenous vein. The total volume of local anaesthetic used should not exceed 4 ml and caution should be taken to avoid intravascular injection or trauma to the vein.

Further Reading

Kopka A, Serpell MG. Distal nerve blocks of the lower limb. *Continuing Education in Anaesthesia Critical Care and Pain*. 2005; 5(5): 166–170.

Levy D, McEwen A. Ultrasound-guided popliteal block. *WFSA Anaesthesia Tutorial of the Week* 2019; 401.

4.2.6 Lumbar Plexus, Femoral and Sciatic Nerve Blocks – Rosie Trumper and Jonathan J Gatward

Could you describe the anatomy of the lumbar plexus?

The easiest way to answer anatomy questions like this is to be able to draw a diagram. A simple line drawing of the lumbar plexus can be easily learnt and reproduced (see Figure 4.2.6.1). A picture paints a thousand words!

The lumbar plexus is located within the posterior third of the psoas major muscle, or between psoas and quadratus lumborum muscles. It arises from the anterior primary rami of L1 to L4. There is sometimes a contribution from T12.

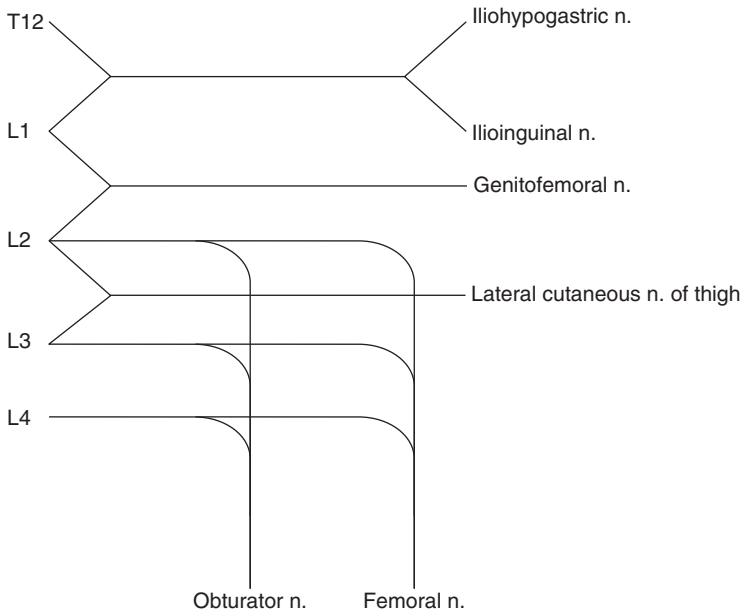


Figure 4.2.6.1 The lumbar plexus

L1 divides into the iliohypogastric and ilioinguinal nerves, and gives a contribution to the genitofemoral nerve.

L2 contributes the remainder of the genitofemoral nerve, and forms the lateral cutaneous nerve of the thigh with L3.

The obturator and femoral nerves are both formed from branches from L2 to 4.

When might we want to perform a lumbar plexus block?

We can use the lumbar plexus block for anaesthesia and post-op analgesia for surgery involving the hip, thigh, or upper leg or after traumatic injury. We can use it in conjunction with a sciatic nerve or sacral plexus block for anaesthesia and analgesia of the leg. In a chronic pain setting, it can be used to treat cancer pain from the hip or upper femur. The sympathetic block achieved can also be useful in the treatment of ischaemic pain and complex regional pain syndrome.

How can the lumbar plexus be blocked? Do you know of any approaches?

The lumbar plexus can be blocked with a posterior approach (the 'psoas compartment block'), and two inferior approaches: the '3 in 1' block and the 'fascia iliaca' block.

Can you describe one of these approaches using a landmark technique?

You are unlikely to be asked to describe all three, so just learn the one that you are most familiar with, and, ideally that you have actually performed. They are all included for the sake of completeness.

For the **psoas compartment block**, the patient is in the lateral position, with the side to be blocked uppermost. The insertion point is the intersection of two perpendicular lines, one running parallel to the spinous processes at the level of the posterior superior

iliac spine and one joining the iliac crests. I use a nerve stimulator and a 100 mm insulated needle inserted at 90 degrees to the skin, aiming slightly caudally. The endpoint is quadriceps contraction, which should occur at a depth of about 8 to 10 cm. An alternative is the loss of resistance technique using a long Touhy needle. You can walk the needle off the inferior surface of the L4 transverse process, and then you get loss of resistance after a further 0.5 to 1 cm.

The '**3 in 1**' block aims to block the *femoral nerve*, the *obturator nerve* and the *lateral cutaneous nerve of the thigh* using a low anterior approach. The patient is in the supine position. The insertion point is 1 cm lateral to the femoral pulse and 2 cm below the inguinal ligament. I use a nerve stimulator and a 50 mm insulated needle, inserted at 45 degrees to the skin aiming proximally and parallel to the femoral artery. The endpoint is quadriceps contraction, which should occur at a depth of 30 to 50 mm. When the local anaesthetic is injected, you can apply distal pressure to help encourage cephalad spread. I usually block the lateral cutaneous nerve of the thigh separately by injecting 10 ml of local anaesthetic beneath the fascia lata at a point 2 cm medial and 2 cm inferior to the anterior superior iliac spine.

The **fascia iliacus block** is convenient because it does not require a nerve stimulator and can be done in the paralysed, anaesthetised patient. It may also be more effective at covering the lateral cutaneous nerve of the thigh than the '3 in 1' block. The patient is in the supine position. The insertion point is 1 cm inferior to the junction of the lateral and middle thirds of a line joining the pubic tubercle and the anterior superior iliac spine. I use either a 16-gauge or 18-gauge Tuohy needle or a blunt nerve stimulator needle. I insert the needle perpendicular to the skin and after penetrating the skin, advance until I feel two more 'pops' as I puncture fascia lata and fascia iliaca. I then inject 30 to 40 ml of local anaesthetic. If using an epidural set, a catheter can be passed, leaving about 3–5 cm in the fascial compartment for continuous infusion of local anaesthetic.

Can you describe the ultrasound-guided lumbar plexus block?

The easiest way to answer anatomy questions like this is to be able to draw a diagram. A simple line drawing of the ultrasound image obtained for performing a lumbar plexus block can be easily learnt and reproduced (see Figure 4.2.6.2).

The use of ultrasound guidance has become a popular technique, increasing the effectiveness, and reducing complications seen with lumbar plexus blocks. Ultrasound can be used in the various approaches (the psoas compartment, 3 in 1 and fascia iliacus approaches), with or without a nerve stimulator. I will describe the ultrasound-guided 'shamrock method' used in the psoas compartment block.

With the patient in the lateral position, the curved array transducer is placed approximately 4 cm lateral to the midline, at the intervertebral space at the level of the iliac crests. With the transducer aimed medially, the 'shamrock' sign can be seen – referring to the appearance of the quadratus lumborum, erector spinae and psoas major muscles. From this position, the transducer is tilted slightly caudally until the L3 transverse process disappears. This is the final ultrasound probe position for the block, allowing an in-plane approach whereby the needle is advanced postero-anteriorly towards the lumbar plexus. Needle insertion is in the same location as described in the psoas compartment block above (again using the landmark technique). The use of a nerve stimulator with production of quadriceps femoris twitch supports correct placement prior to injection.

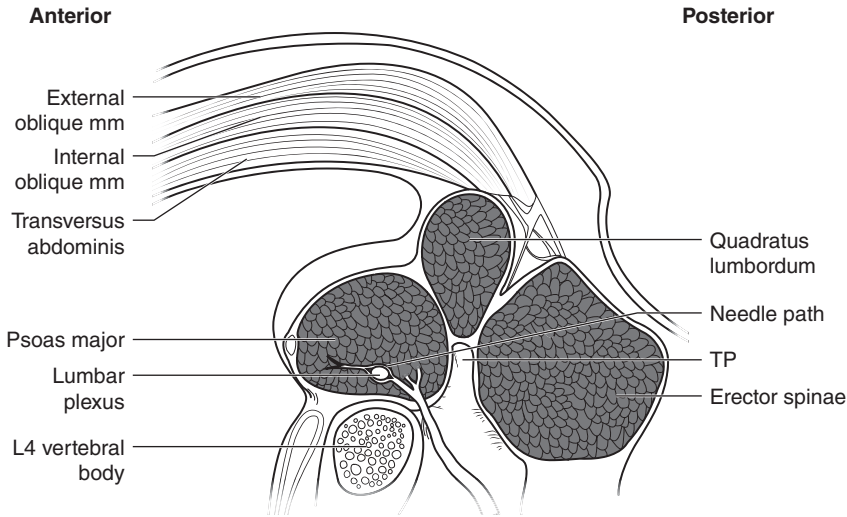


Figure 4.2.6.2 Anatomy and needle path for ultrasound-guided lumbar plexus block.

How does the lumbar sympathetic block differ from the lumbar plexus block?

You've probably never performed a lumbar sympathetic block before. Just be familiar with a method, its complications and the safety measures that are employed when performing the block. The lumbar sympathetic chains lie on the anterolateral aspect of the lumbar vertebrae, whereas the somatic nerves lie posterior and lateral to the psoas muscle and fascia. The anterior relations of the sympathetic chains are the aorta on the left and the inferior vena cava on the right. The aim is to place local anaesthetic solution around the lumbar sympathetic chain from the second to fourth lumbar vertebrae. This can either be achieved by a single injection at L3, or three separate injections at L2, L3 and L4. This block is used in the treatment of a variety of conditions, including lower limb ischaemia, complex regional pain syndrome of the lower limb, phantom limb pain and renal or urogenital pain.

How is the block performed?

The patient is positioned laterally with the side to be blocked uppermost. A point is marked 8 cm lateral to the midpoint of the spinous process of the desired lumbar vertebra. The block is done with either image intensifier or ultrasound guidance. After local infiltration, a 12 cm, 22-gauge needle is inserted at 45 degrees to the skin, aiming medially towards the body of the vertebra.

The body lies at a depth of about 8 cm. If bone is encountered at 4–5 cm, it is likely to be a transverse process, in which case the needle is redirected cranially or caudally to get past it. When the needle has come into contact with the body of the vertebra, it is redirected to pass just anterolaterally. You might feel a loss of resistance as the psoas fascia is penetrated. It is really important to aspirate for blood or cerebrospinal fluid, then a test dose of local anaesthetic mixed with radiographic contrast is injected. This should show a band of contrast along the vertebral column, covering the relevant lumbar vertebral levels. If contrast dissipates quickly, the needle may be in a vessel.

Otherwise, you may see contrast spreading into the retroperitoneal compartment or into the psoas muscle. Lateral and anteroposterior images should be obtained to make sure the needle is in the right place.

What specific complications of this block do you know?

Local anaesthetic toxicity can be caused by inadvertent intravascular injection into the aorta or inferior vena cava. Intrathecal injection can occur, which would cause a profound motor block or permanent paralysis if neurolytic agents were used. The block can also cause profound hypotension, so intravenous access, full monitoring and access to resuscitation equipment is mandatory. Other complications are ureteric damage and ejaculatory failure. Also, post-sympathectomy neuropathic pain can occur with neurolytic techniques.

Can you describe the anatomical course of the sciatic nerve?

While revising, practise drawing simple line diagrams of the sacral plexus, the sciatic nerve, and its divisions, adding in some of its relations and landmarks to help you remember its course (see Figures 4.2.6.3, 4.2.6.4).

The sciatic nerve arises from the sacral plexus, which is formed by the nerve roots of L4, L5 and S1–3. It passes anterior to the piriformis muscle and exits the pelvis through the greater sciatic notch. It passes into the posterior thigh between the ischial tuberosity and the greater trochanter of the femur. It then passes posterior to the femur, before dividing into the tibial and common peroneal nerves in the popliteal fossa. The tibial nerve passes medially and divides into the sural and posterior tibial nerves. The common peroneal nerve passes laterally and divides into the deep and superficial plantar nerves.

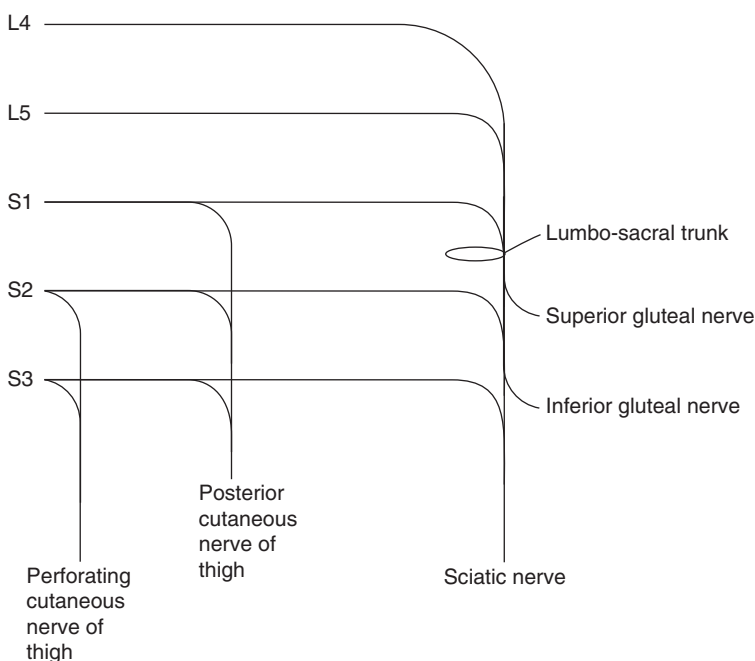


Figure 4.2.6.3 The sacral plexus.

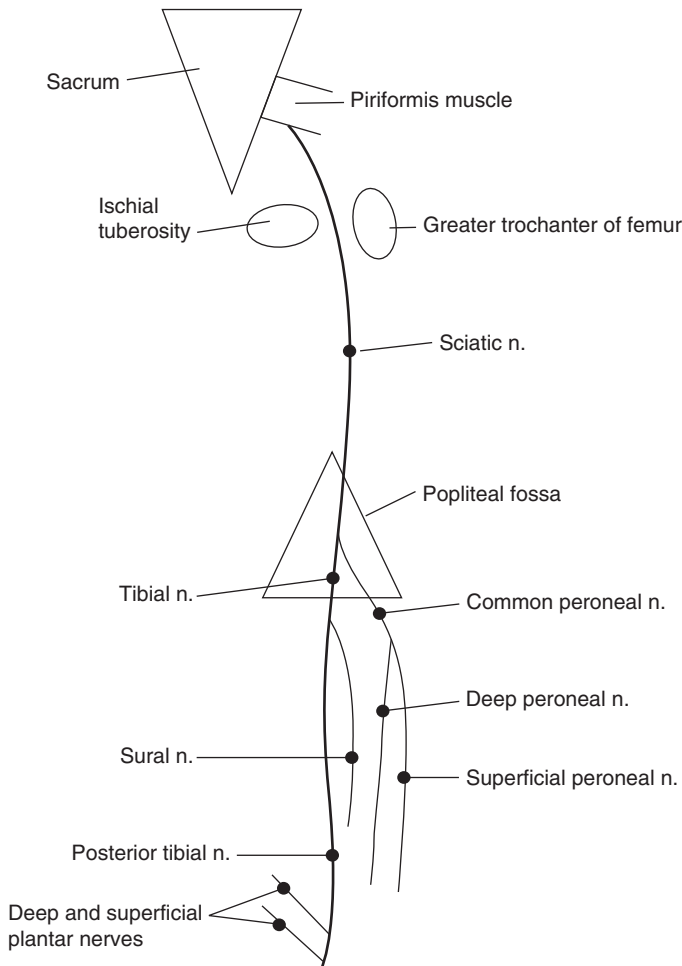


Figure 4.2.6.4 The sciatic nerve.

Can you describe the motor and sensory functions of the sciatic nerve?

The sciatic nerve supplies motor innervation to:

- Hamstrings
- Soleus
- Gastrocnemius
- Peroneal muscles
- Tibialis anterior
- Intrinsic muscles of the foot.

It supplies sensory innervation to:

- Hip and knee joints
- Most of the leg except the medial knee and calf, which are supplied by the saphenous nerve, a branch of the femoral nerve.

When might we want to perform a sciatic nerve block?

The sciatic nerve block can be used alone to provide postoperative analgesia for ankle and foot surgery, or analgesia for fractures of the lower leg. In combination with a femoral nerve block, it can be used for analgesia for knee and lower leg surgery and amputations. In the chronic pain setting, it can be used for ischaemic pain and in CRPS.

What are the different approaches to the sciatic nerve block?

You are unlikely to be asked to describe more than one or two approaches. Again, they are all given for the sake of completeness. Draw a diagram if it helps you remember the anatomy!

There are three posterior, one anterior and one lateral approach to the sciatic nerve. Ultrasound guidance is useful in each case, improving success rate and reducing complications.

Labat's posterior approach is performed with the patient in the lateral position with the upper leg flexed at 90 degrees at the hip and knee. I draw two lines; one from the posterior superior iliac spine to the greater trochanter and the other from the greater trochanter to the sacral hiatus. I then drop a third line at 90 degrees from the upper line. The insertion point is where this perpendicular line hits my lower line. I use a 100 mm needle, inserted at 90 degrees. The endpoint I am looking for is plantar flexion, which I would expect at about 6 cm.

Another **posterior approach** occurs with the patient in the supine position, with the leg flexed at 90 degrees at the hip and knee. I draw a line from the greater trochanter to the ischial tuberosity. The midpoint of this line is the insertion point. I use a 100 mm needle, inserted at 90 degrees. The endpoint I am looking for is plantar flexion, which I would expect at about 6 cm.

The third posterior approach is the **popliteal fossa block**. This can be performed with the patient either prone with the leg straight, or supine with the hip and knee flexed at 90 degrees. The insertion point is 7 to 8 cm proximal, and 1 cm lateral to the midpoint of the skin crease of the knee. I use a 50 mm needle, inserted at 45 degrees proximally. The endpoint I am looking for is plantar flexion. However, the nerve may have already divided at this point, so I may get dorsiflexion caused by stimulation of the common peroneal nerve. If this occurs, I deposit half the local at this point and then redirect medially to locate the tibial nerve, looking for plantar flexion. I would deposit the rest of the local there. With the use of ultrasound, I can bring the probe cephalad from the popliteal fossa to confirm the point at which the sciatic nerve divides and deliver local anaesthetic above this division to ensure adequate blockade of the tibial and common peroneal nerves.

The **anterior approach** to the sciatic nerve is performed with the patient in the supine position. I draw a line connecting the anterior superior iliac spine and the pubic tubercle. I then divide this into thirds, and drop a perpendicular line from the junction of the middle and medial thirds of this line. The insertion point is where this line meets a parallel line at the level of the greater trochanter. I use a 100 mm needle, inserted at 90 degrees. The endpoint I am looking for is plantar flexion, which I would expect at about 8 to 10 cm. If I hit bone, I redirect medially.

The **lateral approach** is performed with the patient in the supine position. A line is drawn down the thigh from the posterior border of the greater trochanter. The insertion point is halfway between the knee and the greater trochanter. I use a 100 mm needle, inserted at 90 degrees. The endpoint I am looking for is plantar flexion, which I would expect at about 8 to 10 cm. If I hit bone, I redirect posteriorly.

What general safety measures do you employ when performing a regional block?

I take a full medical and anaesthetic history and ask about drug allergies. I secure intravenous access, and ensure that full airway and cardiovascular resuscitation equipment is immediately available. Patients should be fully monitored. I use a strict aseptic technique, with antiseptic solution, sterile gloves and sterile, single-use needles. Aspiration prior to injection reduces the risk of inadvertent intravascular or intrathecal injection of local anaesthetic and additives.

In general, what methods are available to identify nerves when performing regional blocks?

Nerves can be identified in several ways:

- The most basic way is to use anatomical landmarks to find the insertion point. You can then insert the needle to a given depth, such as in the paravertebral block, or there may be an endpoint, such as a 'pop' through fascia. With practice, the feeling of injecting a solution around a nerve and between fascial planes can be appreciated. This differs, sometimes quite subtly, from the high pressure required to inject solution intramuscularly and the low pressure of injection into a vessel.
- Some anaesthetists elicit paraesthesia in the distribution of the nerve concerned, though there is a risk of nerve damage.
- Loss of resistance techniques can be used, for example in epidural insertion, psoas compartment and paravertebral blocks. This can be the loss of resistance to the injection of air or saline.
- Another method is to transfix an artery lying within a nerve sheath such as the axillary artery, in axillary nerve blockade.
- Most anaesthetists now use nerve stimulation techniques, either with a non-invasive probe to help identify nerves, or with special insulated needles.
- Ultrasound-guided techniques are gaining popularity and may be combined with other techniques to try to improve accuracy. This allows visualisation of the nerve, surrounding structures and the block needle in real time. The spread of local anaesthetic around the nerve can also be seen and predicts a successful block.

What are the basic principles behind nerve stimulators? How do they work?

Electrical impulses are delivered via a needle to elicit either paraesthesiae or the muscle movement associated with the nerve concerned. They can help to reduce the incidence of nerve damage because the aim of the technique is to get the tip of the needle very close to the nerve without touching it. They still require a thorough knowledge of the relevant anatomy.

Talk us through how you would use a peripheral nerve stimulator.

We'll assume you have IV access, monitoring, skilled assistance, and there are no contraindications.

I would connect the positive lead to an ECG electrode at least 20 cm from the needle insertion site and the negative lead to an insulated nerve stimulator needle of the required length. I remember which lead is which as I can remember that the

patient is positive and the needle is negative. I set the duration of impulse at 100 msec or less and the frequency of impulse at 1–2 Hz. To start with, I set the delivered current at 1–2 mA.

I then prepare the area, inject some local anaesthetic into the skin with an orange needle, wait for it to work and then insert the stimulator needle. I look for synchronous muscle movement in the desired distribution and when I get it, I reduce the current. Movement should still be present at 0.5 mA, but should disappear at 0.2 mA, or the needle might be within the nerve. It is really important to aspirate at this point to check for intravascular placement. I then inject 1 ml of local anaesthetic, at which point the motor response should disappear as the nerve is displaced away from the needle tip.

Injection should be painless and without resistance. If not, I would consider repositioning. I usually get an assistant to inject the local and get them to aspirate after each 5 ml increment. This is to check that the needle tip has not migrated into a blood vessel. To further increase the safety of this technique, ultrasound can be used to visualise the correct needle placement and spread of injectate in real time.

Could you compare and contrast lidocaine and bupivacaine when used for nerve blockade?

Both are amide local anaesthetic solutions commonly used in nerve blocks.

Lidocaine has a rapid onset and short duration of action. It causes vasodilatation at the site of injection, and so the addition of adrenaline 1:200000 slows its systemic absorption and prolongs the duration of block. The maximum dose is 3 mg/kg or 7 mg/kg if adrenaline is added.

Bupivacaine has a slower onset of action and longer duration of action. It lasts two to three times longer than lidocaine. It does not cause vasodilatation, so the addition of adrenaline has no effect on its systemic absorption or its duration of action. The maximum dose is 2 mg/kg. Levobupivacaine, the S-enantiomer of bupivacaine, is less cardiotoxic. Its maximum dose is also 2 mg/kg.

What do you know about ropivacaine?

It has an onset of action similar to bupivacaine, but causes some vasoconstriction that may prolong its duration of action. Like bupivacaine, its maximum dose is 2 mg/kg. It is marketed as causing less motor block and cardiotoxicity than bupivacaine, but this may just be due to lower potency.

4.2.7 Local Anaesthetic Toxicity – Andrew P Georgiou

What is a local anaesthetic agent?

You need to know an off-pat definition here in order to get the examiner on your side from the start.

A local anaesthetic agent is one that reversibly blocks neural transmission beyond its point of application, when applied locally.

All local anaesthetic agents are weak bases and can be subdivided into esters and amides, according to the chemical linkage between the hydrophobic aromatic ring structure and the hydrophilic chain.

Can you give me some examples of esters and amides?

- Examples of amides include lidocaine, bupivacaine and prilocaine.
- Examples of esters include cocaine, tetracaine and amethocaine.

Amides are more commonly used in clinical practice due to their increased stability, ease of heat sterilisation and longer shelf life.

How do local anaesthetic agents work?

Local anaesthetic agents work by blocking the voltage-gated sodium channel from the cytosolic aspect of the receptor, that is from the inside of the cell. Inactivation of the sodium channel prevents the upstroke of the action potential and so inhibits depolarisation and therefore transmission of nerve impulses.

The drug accesses the receptor by crossing the neural membrane in its unionised form. It then becomes ionised in the relative acidic environment of the cytoplasm and it is the ionised form that is responsible for receptor blockade.

A secondary mechanism of action relies on disruption of impulse transduction due to the presence of unionised agent in the nerve cell membrane.

What factors determine the potency, the speed of onset and the duration of action of a local anaesthetic?

Potency is dependent on how lipid soluble the drug is, the more lipid soluble it is, the more potent it becomes.

The onset of action is related to the drug's pKa. A drug with a lower pKa will have a greater percentage present in the unionised form, which allows more rapid passage through the nerve cell membrane and a faster onset of action. So, for example, lidocaine has a pKa of 7.9 and has a more rapid onset of action than bupivacaine which has a pKa of 8.1.

The duration of action is dependent on how protein bound the drug is. If a drug is highly protein bound, like bupivacaine, it will have a long duration of action.

What factors place a patient at risk of local anaesthetic toxicity?

Local anaesthetic toxicity results from the acquisition of a high or rapidly rising plasma local anaesthetic concentration. The factors which place a patient at risk of this situation developing are:

Firstly, the dose administered and the speed of injection, as large, rapidly administered doses raise the risk of high plasma concentrations developing.

Secondly, inadvertent vascular injection, for example through an epidural catheter, inadvertently placed into a vein in Batson's plexus, resulting in direct administration of local anaesthetic into the circulation.

Similarly inadvertent intrathecal injection will also produce toxic effects as an inappropriate intrathecal dose is likely to be administered.

Thirdly, the site of administration, as this affects the rate of absorption of local anaesthetic into the bloodstream. So, for example, local anaesthetic is more readily absorbed into the bloodstream when it is administered via the intercostal or caudal site, compared to the subcutaneous or brachial plexus site. The performance of a pudendal nerve block (in obstetrics) is also highly likely to cause systemic toxicity due to the adjacent rich blood supply.

Related to this is the presence of vasoconstrictor in the injected solution. For example, adrenaline may be co-administered with local anaesthetic agents producing localised vasoconstriction and limiting the absorption of local anaesthetic agent into the bloodstream. Adrenaline added to lidocaine increases the safe dose which may be given from 3–6 mg/kg for this reason.

Fourthly, the type of local anaesthetic used as some types of local anaesthetic are inherently more toxic than others. For example, racemic bupivacaine is inherently more toxic than its pure S-enantiomer levobupivacaine. This is due to a reduction in the avidity of binding of the S-enantiomer to the sodium channels of the myocardium, and therefore less risk of the myocardial depressant effects of a toxic dose.

Fifthly patient heterogeneity and comorbidity has an important bearing on the risks of toxicity. Patients with cardiac or hepatic disease and those who are critically unwell or pregnant as well as neonates and young infants are particularly at risk due to alterations in absorption, distribution, metabolism and excretion. These patients also have changes in their protein binding capacity, particularly to alpha-1 acid glycoprotein (where the levels are reduced), and therefore may well have increased free drug concentration, raising the risk of toxicity. In these situations, the dose administered should be reduced.

Finally, the acid–base status of the patient has a bearing on their risk of toxicity. Avoiding acidosis (so for example, ensuring patients are adequately ventilated) will reduce the risk of toxicity occurring.

Some patients are particularly at risk of a specific form of toxicity related to one of the amide local anaesthetic agents. Can you think of what that may be?

Prilocaine is metabolised to O-toluidine. Infants and those with methaemoglobinaemia reductase deficiency are poorly able to metabolise O-toluidine and may develop methaemoglobinaemia, which results from the oxidation of the iron atom in haem from its ferrous to its ferric state. This produces a blue discoloration of the skin, which can produce errors in pulse oximeter analysis. Pulse oximeters may falsely read 85% leading the anaesthetist to believe the patient is hypoxic, where this may not be the case.

The oxygen-carrying capacity of haemoglobin is reduced by the transformation in haem and in extreme cases this may result in tissue hypoxia.

How is this condition treated?

It can be treated with methylene blue at a dose of 1 mg/kg over 5 minutes.

It is important to remember, however, that prilocaine is relatively less toxic compared with bupivacaine, especially on the heart. This is why it is used for intravenous regional anaesthesia.

Let's get back now to local anaesthetic toxicity in general. What are the symptoms and signs of local anaesthetic toxicity?

Most people can rattle off a list in answer to this question, but if you can bring in some basic science to your answer it looks like you really understand the concepts, making you a stronger candidate.

Local anaesthetic toxicity presents initially with central nervous system disturbance followed by cardiovascular system disturbance.

Toxic levels of local anaesthetic initially depress the depressant effects of the central nervous system, such that excitatory effects are initially seen.

These include headache, perioral tingling, tinnitus, vertigo, disorientation, visual and auditory disturbances, and a sense of impending doom. Shivering and tremors may be seen.

As the toxic effect progresses, both the depressant and the excitatory effects of the central nervous system are depressed. This may then lead to slurred speech, loss of consciousness and seizure activity, often manifesting as a grand mal type fit.

Escalating toxicity then results in respiratory depression and arrest and cardiovascular disturbances. This is initially hypotension from direct cardiac depression and vasodilation, followed by cardiac arrest with any number of dysrhythmias intervening. Sinus bradycardia, sinus arrest and resistant VF would be common examples.

What do you understand by the term 'toxicity ratio'?

The toxicity ratio is the ratio between the plasma level of local anaesthetic required to produce cardiovascular symptoms to that required to produce central nervous system symptoms. It is expressed as a ratio for each local anaesthetic agent and gives an idea of the window between the onset of central nervous system symptoms and the onset of cardiovascular symptoms.

For example, the toxicity ratio for lidocaine is 7 and for bupivacaine is 3, but for levobupivacaine is 5. This means that after CNS symptoms occur you need seven times that plasma level of local anaesthetic to get cardiovascular symptoms with lidocaine, but only three times that plasma level to get cardiovascular symptoms with bupivacaine. In practical terms this means that the onset of central nervous system symptoms in bupivacaine toxicity means that cardiovascular symptoms are soon to follow.

So why is bupivacaine more toxic than lidocaine?

Bupivacaine is 4–16 times more cardiotoxic than lidocaine due to its increased protein binding and the avidity with which it binds myocardial tissue. It dissociates from myocardial sodium channels 10 times less readily than lidocaine, producing a prolonged, refractory blockade. Reversal of this binding can be particularly difficult, especially in the face of cardiopulmonary arrest when hypoxia and acidosis may complicate the picture.

How would you manage a 28-year-old primigravida who has collapsed after complaining of tinnitus and vertigo following an epidural top-up of 20 ml of 0.5% levobupivacaine?

She is scheduled for a category 2 caesarean section. This is an emergency; the factors which pose the greatest threat to the mother's life should be managed first and without delay.

I would call for help immediately while placing the woman in a left lateral tilt of at least 15 degrees.

I would start by ensuring a patent airway and administer high flow oxygen via reservoir mask. Should the airway be compromised, I would secure it with a cuffed endotracheal tube. While assessing the airway I would palpate a central pulse to exclude cardiac arrest, before going on to assess the breathing.

There is no palpable pulse. How would you proceed?

I would dedicate a member of staff to put out a cardiac arrest call and ask them on their return to bring the resuscitation trolley and more members of staff to assist. I would then allocate another member of staff to bring the 20% intra-lipid.

In the meantime I would start advanced life support according to the Advanced Life Support guidelines. This involves cardiopulmonary resuscitation in the ratio of 30 chest compressions to 2 ventilations, or asynchronous compressions once the trachea is intubated. I would assume the role of cardiac arrest team leader and delegate team members to appropriate roles.

Discovery of the underlying rhythm is then essential to allow stratification to the VF/pulseless VT algorithm or to the asystole/PEA algorithm.

You mentioned intra-lipid. How is this administered in this setting?

On arrival of the 20% intra-lipid, I would immediately deliver a 1.5 mg/kg bolus IV and start an infusion of 0.25 mg/kg/min. The bolus injection may be repeated twice at 5-minute intervals. At 5 minutes after the third bolus of intra-lipid the infusion rate should be increased to 0.5 mg/kg/min.

It is worth bearing in mind that cardiac arrest due to local anaesthetic toxicity may be prolonged and refractory to treatment so I would persist with the resuscitation for over an hour if needed.

Although standard ALS protocols should be followed throughout, consideration should be given to the use of cardiopulmonary bypass where available, as the situation is potentially salvageable with prolonged life support.

Is there anything else that you may consider in this situation?

If after 5 minutes of CPR there has been no return of spontaneous circulation then a perimortem caesarean section should be undertaken, in order to optimise the efficacy of CPR and the chance of maternal survival.

Ventricular fibrillation should be treated according to standard ALS protocols. Magnesium should be considered at an early stage. Should the VF prove refractory to standard treatment and intra-lipid, then bretylium may be considered. It is not part of the standard resuscitation guidelines but in this situation, there is a small chance it may be of benefit.

You mentioned bretylium. How does this drug work and what dose would you use?

Bretylium acts by inhibiting release of noradrenaline at the sympathetic nervous system and is administered by rapid IV injection at a dose of 5 mg/kg. If VF persists the dose may be increased to 10 mg/kg and given at 1–2 hour intervals. Bretylium is not commonly used and so its acquisition is unlikely to be rapid; hence the importance of all other aspects of resuscitation in this case.

Where is intra-lipid kept in your hospital?

You should know the answer to this, and if you don't, make it look like you do!

Intra-lipid is kept in areas where potentially toxic doses of local anaesthetic are administered. It is kept on the resuscitation trolley in theatre, on the resuscitation trolley

on delivery suite, in the bag that the ICU registrar brings to arrest calls and it is available in the pharmacy store cupboard in theatres and from pharmacy.

What is the actual mechanism of local anaesthetic toxicity?

Local anaesthetic toxicity results from several processes, most commonly binding of the anaesthetic agent to the cytosolic aspect of the voltage-gated sodium channel resulting in sodium-channel blockade. Bupivacaine, one of the most toxic local anaesthetic agents, also interferes with almost all of the metabotropic and ionotropic cell transduction mechanisms studied. It also interferes with oxidative phosphorylation, perhaps most importantly the metabolism of carnitine, which is thought to be essential for the transport of fatty acids into the mitochondria. The transport of fatty acids into mitochondria is essential for the generation of ATP and so bupivacaine, in disrupting this process, limits the ability of cells to produce the energy they need to function normally. This disruption in ATP generation is particularly apparent in energy demanding cells, such as cardiac myocytes.

How does intra-lipid alter this process?

If you get a question like this, don't panic, it means you are doing well!

Twenty per cent intra-lipid has an avidity for local anaesthetic agents which may prevent the agent reaching the cell if administered reasonably soon after the toxic dose. It may have a potential role in drawing the agent out of the cell and removing it from its inhibitory action on metabotropic and ionotropic signalling, and restoring the action of carnitine on fatty acid transport in mitochondria.

This effect results in an apparent shift in the dose–response curve resulting in a higher plasma concentration of local anaesthetic agent required to induce asystole. Weinberg and colleagues, amongst others, have explored this in the experimental setting of rats and dogs, and users of intra-lipid are encouraged to report cases to intra-lipid.org where case series are being compiled.

Further Reading

Cave G, Harrop-Griffiths W, Harvey M.
Guidelines for the management of severe
local anaesthetic. Toxicity. *The Association
of Anaesthetists of Great Britain and*

Ireland. 2010; [www.aagbi.org/publications/
guidelines/docs/latoxicity07.pdf](http://www.aagbi.org/publications/guidelines/docs/latoxicity07.pdf).

Weinberg G. LipidRescue: Resuscitation for
cardiac toxicity. [http://lipidrescue.
squarespace.com/](http://lipidrescue.squarespace.com/).

Pain

4.3.1 Pain Pathways – Michael B Clarke

Can you define pain?

There is a standard definition in answer to this question that is worth knowing.

Pain is an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described in terms of such damage.

Can you describe the different types of pain receptor?

Nociceptors are the sensory receptor for pain and are widespread. They are responsive to different types of stimuli; mechanical, thermal or chemical. Stimulation of these nociceptors results in propagation of an impulse to the spinal cord.

The primary afferent fibres involved in this impulse propagation can be divided into two classes:

1) A δ fibres

- Myelinated
- 2 to 5 μm in diameter
- Conduct rapidly (6 to 30 m/s)
- Responsible for localised, sharp pain sensations
- Provoke the withdrawal reflex.

2) C-fibres:

- Unmyelinated
- Smaller diameter
- Conduct less rapidly (0.5 to 2 m/s)
- Responsible for poorly localised, dull, burning-type pain.

How does stimulation of these nociceptors cause us to sense pain?

This question is dealing with the route the pain impulse takes to the somatosensory cortex. The answer is complex but detailed knowledge will not be expected. Just give a simple answer including the basic points and the examiner should be satisfied.

Primary afferents have their cell bodies located in the dorsal root ganglia and synapse with secondary afferents in the dorsal horn. C-fibres tend to enter the dorsal horn laterally, while A δ fibres tend to enter the dorsal horn more medially.

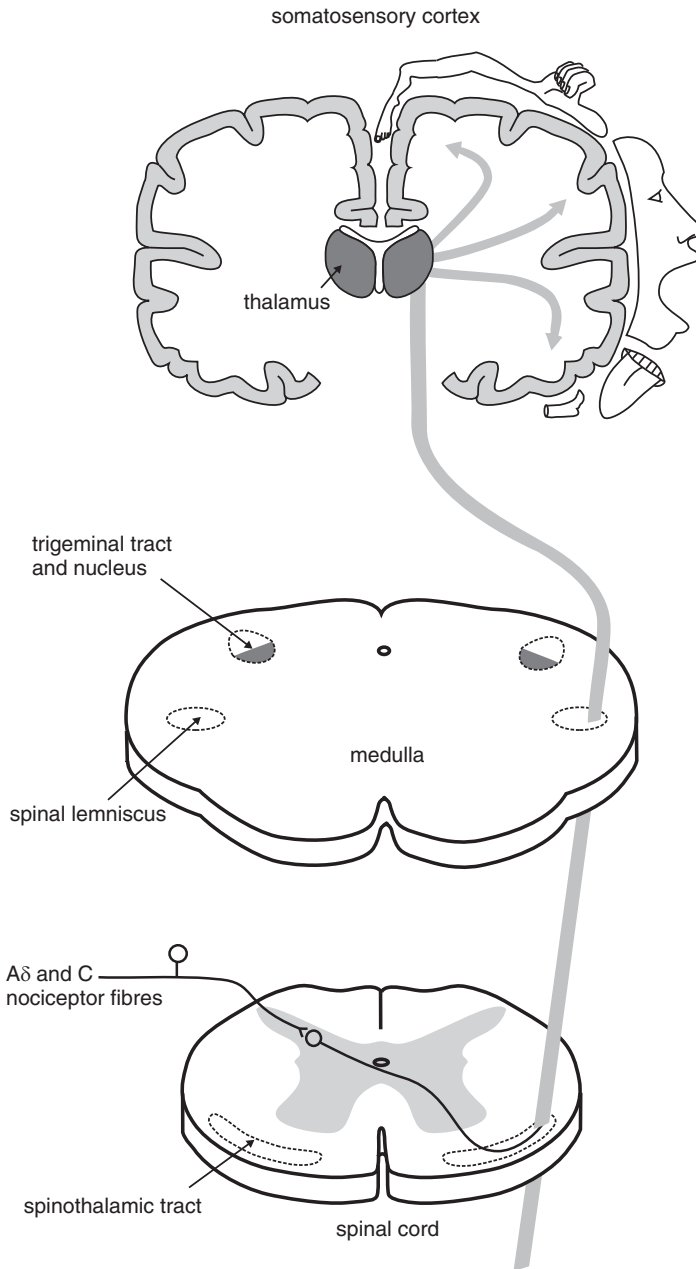


Figure 4.3.1.1 Somatosensory pain pathway.

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The grey matter of the dorsal horn is divided into laminae that run the length of the spinal cord. Aδ fibres synapse with cells principally in laminae I and V. C-fibres synapse with cells in laminae II and III and the substantia gelatinosa (Figure 4.3.1.1).

There are two main classes of secondary afferents within the dorsal horn associated with further processing of the noxious stimulus. The first class is 'nociceptive specific' or 'high threshold'. These are located within the superficial laminae and respond to noxious stimuli only. The second class is termed 'wide dynamic range' or 'convergent'. These are located in the deeper laminae and respond to both noxious and non-noxious stimuli. Wide dynamic range neurons will fire in proportion to the intensity of the stimulus. Normally they do not signal pain; however, if they become sensitised and their activity exceeds a certain threshold, a normally non-noxious stimulus will be perceived as being painful; this is called allodynia.

From the dorsal horn, the main nociceptive pathway is the spinothalamic tract. Secondary afferents cross the spinal cord and then ascend as the lateral spinothalamic tract. Afferents from here will terminate in various structures throughout the brainstem and thalamus, and then on to the somatosensory cortex. Areas of the cortex involved in pain processing include the post central gyrus, the Sylvian fissure and the cingulate gyrus.

How is it possible then that we can receive a tissue injury and yet not experience pain, for example soldiers in battle who are unaware they have been injured?

This question concerns the modulation of pain signals and the perception of pain. A simple example of this is gate control in the dorsal horn and a diagram showing primary and secondary afferents with inhibitory interneurons and descending projections acting on interneurons will help you quickly answer this (Figure 4.3.1.2).

The modulation of the nociceptive signal can occur at all levels of the pain pathway. The output from the dorsal horn of the spinal cord depends on the input from the periphery, regulation by interneurons and descending projections from the brain.

Descending projections from the periaqueductal grey matter and the locus coeruleus of the midbrain play an important role. The periaqueductal grey matter receives input from the frontal cortex, the limbic system, the thalamus and the hypothalamus. It is

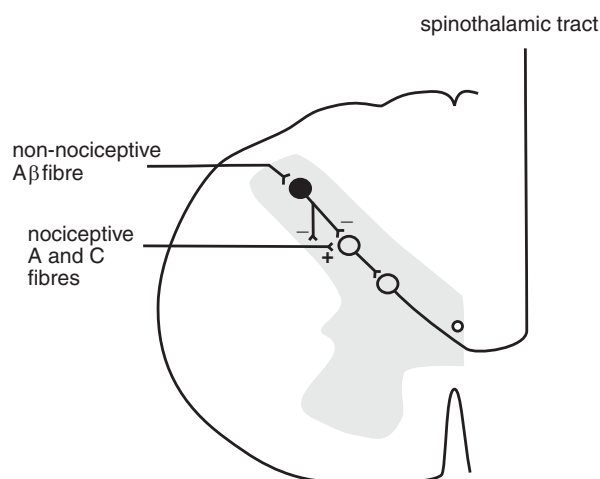


Figure 4.3.1.2 Gate mechanism. Reproduced with permission from Smith, T., Pinnock, C. and Lin, T. 2009. *Fundamentals of Anaesthesia*. Cambridge: Cambridge University Press. © Cambridge University Press 2009.

involved in the production of endogenous opioids and its projections stimulate inhibitory interneurons in the dorsal horn.

A β afferents carry touch sensation from the periphery and can also stimulate inhibitory interneurons within the dorsal horn. The presence of interneurons means that the nociceptive signal can be 'gated' and the relative activity of the A β , A δ and C-fibres, plus the contribution of descending projections from the brain will either open or close the gate. If nociceptive input is greater than A β input, the gate will open and the nociceptive impulse will continue; if the A β input exceeds the nociceptive input, the gate is closed and the nociceptive signal is stopped (Figure 4.3.1.2). This is why rubbing the skin can make a painful area 'feel better'.

What is visceral pain and how does it differ from peripheral pain?

Visceral pain describes pain originating from the internal organs. It is poorly localised and associated with nausea and autonomic disturbance. It occurs with distension and ischaemia rather than thermal or mechanical trauma.

Nociceptors are present in the viscera but are less dense than in somatic structures; hence the poor localisation of pain. The cell bodies are located in the dorsal root ganglia and the afferent fibres reach the spinal cord via sympathetic and parasympathetic pathways. Secondary afferents travel in the spinothalamic tracts and project to the same areas of the somatosensory cortex as peripheral secondary afferents.

What is referred pain?

This is where pain originating from a viscus is felt in a somatic structure at a distance from the viscus, for example, diaphragmatic irritation causing shoulder-tip pain. It occurs because visceral and peripheral primary afferents converge upon the same secondary afferents and the brain cannot discern whether the pain is coming from the organ or the periphery.

If I cut my hand, why does the area around the injury also become red and painful?

A cut to the skin will not only activate nociceptors but will also release a number of inflammatory peptides, these include substance P and neurokinin A. Release of these chemicals causes local vasodilatation giving rise to the area of redness. They also stimulate the release of further inflammatory mediators from mast cells and platelets. These substances include histamine and serotonin. These then act to sensitise surrounding nociceptors, a process known as peripheral sensitisation. Now, pressure on the area immediately around the cut that would normally cause only slight pain or no pain at all will produce an exaggerated response and pain will be experienced. This is known as primary hyperalgesia.

Can sensitisation occur centrally?

Yes, it can. Hyperalgesia in the uninjured skin further removed from the site of injury is called secondary hyperalgesia. Nociceptors in this area are not sensitised and so the mechanism for this is believed to be due to changes in the dorsal horn of the spinal cord; this is called central sensitisation.

What does this tell us about pain and the central nervous system?

The important point that this highlights, is that in the presence of pain, 'plasticity' exists within the central nervous system. In other words, sustained nociceptive inputs have the ability to alter the functional properties of dorsal horn neurons. It has been shown that a painful stimulus will not only cause a dorsal horn neuron to fire but that the rate of firing will increase with the duration of the stimulus. This is termed 'wind-up' and it is an important part of central sensitisation.

There are other important features of central sensitisation. There is an expansion of the receptive field of neurons, meaning for any given noxious stimulus, more dorsal horn neurons will respond. There is an increase in the size and duration of the response to a noxious stimulus. Therefore, the threshold for stimulation of dorsal horn neurons is decreased, so that normally non-noxious stimuli will activate dorsal horn cells.

Does this have implications for different types of pain?

Yes. It may be important in the development of chronic pain states.

Can you define chronic pain?

Chronic pain is defined as pain without apparent biological value that persists beyond normal tissue healing, usually taken to be 3 months.

Can you classify chronic pain?

Chronic pain can be described as inflammatory, neuropathic or dysfunctional.

Inflammatory pain is produced when tissues are damaged due to trauma or infection.

Neuropathic pain is due to a primary lesion or pathology of the peripheral or central nervous system. Examples include phantom limb pain, nerve entrapment pain, as in carpal tunnel syndrome, peripheral neuropathies secondary to diabetes and alcoholism, and central neuropathies due to spinal cord trauma and stroke.

Dysfunctional pain describes poorly localised pain that is not due to tissue inflammation or nerve damage and includes conditions such as fibromyalgia and irritable bowel syndrome.

Can you describe the pathways that produce chronic pain?

Since many of these pathways have not been elucidated the examiners should not expect too much detail.

It is not fully understood how pain signals can persist over long periods of time. A simple way of describing postulated mechanisms is to look at changes first in the periphery and then centrally.

Following an injury, nerves can become sensitised and discharge at lowered thresholds. C-fibres can develop new adrenergic receptors, which may help to explain the mechanism of sympathetically mediated pain. Damaged nerves also produce ectopic discharges, thought to be due to the formation of dysfunctional sodium channels.

Inflammatory mediators such as substance P and prostaglandins are released from damaged nerves. These can then spread to surrounding nociceptors activating them in turn.

Following a peripheral nerve injury, changes within the central nervous system can persist. This plasticity of the CNS is integral to the development of chronic pain states. Repetitive stimulation of C-fibres can result in the phenomenon of 'wind-up', where the rate of firing of dorsal horn cells will increase with the duration of the stimulus. Repetitive episodes of wind-up may precipitate 'long-term potentiation'. Long-term potentiation is defined as a long-lasting increase in synaptic activity. The NMDA receptor is believed to play an important role in these sensitisation processes.

As well as changes at the spinal cord level, alterations in the descending pathways from the brainstem and higher centres will also play a role in the development of chronic pain states. This is highly complex but will involve the areas involved in the sensory-discriminative aspects of pain such as intensity and location, as well as centres involved in the affective-cognitive aspects of pain including anxiety, emotion and memory.

4.3.2 Neuropathic Pain including Complex Regional Pain Syndrome, Trigeminal Neuralgia and Post-herpetic Neuralgia – Shelley Barnes and Sarah Love-Jones

These topics are well suited to the clinical sciences SOE, but equally they may come up as part of the clinical SOE.

Neuropathic Pain

What is neuropathic pain?

Neuropathic pain is often initiated or caused by a primary lesion or dysfunction in the peripheral or central nervous system. Neuropathic pain can also be called neurogenic pain. Neuropathic pain can be in a region of motor, sensory or autonomic nerve dysfunction. Common descriptions of neuropathic pain include: burning, stabbing, sharp, tingling, numb, electric shock-like and paraesthesia.

Can you give me any examples of neuropathic pain conditions?

There are several types of neuropathic pain conditions routinely seen in pain clinic.

These are:

- Lumbar radicular pain
- Trigeminal neuralgia
- Post-herpetic neuralgia
- Complex regional pain syndrome (CRPS)
- Phantom limb pain
- Diabetic or drug-induced neuropathy
- Central pain syndromes.

Can you describe the clinical features of neuropathic pain?

Neuropathic pain can be associated with spontaneous pain, in the absence of any stimulus. This spontaneous pain may be continuous or paroxysmal. Symptoms vary according to the types of nerves affected.

A sensory neuropathy affects sensation of touch, temperature, pain etc. Resulting features can include allodynia, hyperalgesia, numbness, pins and needles etc. Allodynia (pain after an innocuous stimulus) and hyperalgesia (pain of abnormal severity following a painful stimulus) are evoked pains.

A motor neuropathy may damage nerves that control movement. An autonomic neuropathy may damage nerves that control involuntary processes such as digestion, regulation of blood pressure and bladder function.

How can neuropathic pain be diagnosed?

Primarily with a focussed clinical history and clinical examination.

Specific investigations may be required such as nerve conduction studies, electromyography or nerve biopsy.

How can neuropathic pain be subsequently managed?

The treatment of neuropathic pain is difficult. No individual treatment works for everyone. Often multiple therapies are used simultaneously but it may take trial and error to achieve optimal benefit.

Treatment often commences with the management of possible underlying conditions (e.g. diabetic control, replacing vitamin B12 or stopping any drugs that might be contributing to the pain).

The treatment of neuropathic pain can be divided into five categories:

- Pharmacological therapy
- Intra-articular or epidural injections
- Neuromodulation
- Physiotherapy
- Cognitive behavioural therapy (CBT).

What medications can be used in the treatment of neuropathic pain?

Be systematic in your approach.

Simple analgesics such as paracetamol, non-steroidal anti-inflammatory drugs (NSAIDs) and weak opioids such as codeine should always be tried first and this is usually done by self-medicating or obtaining advice from a GP. These are often ineffective and require adjunctive therapy.

The tricyclic antidepressants such as amitriptyline and nortriptyline are the next class of drugs commonly used in pain clinics to treat neuropathic pain. Tricyclic antidepressants act by blocking the reuptake of monoamines, and it is both the noradrenergic and serotonergic effects that are needed in the treatment of neuropathic pain. It is worth explaining to the patient when prescribing tricyclic antidepressants that their analgesic effect is separate from their antidepressant effect.

The membrane-stabilising drugs such as the anticonvulsants carbamazepine, gabapentin and pregabalin and local anaesthetics such as lidocaine are also used to treat neuropathic pain. It is thought that the mechanism of action of gabapentin and pregabalin is that they bind to the α -2 delta subunit of the voltage-dependent calcium channel and have calcium-channel blocking activity.

Topical treatments used in neuropathic pain include capsaicin cream, which is thought to deplete peptide neurotransmitters in the nerve endings. Lidocaine patches are also used for discrete painful areas. Non-steroidal anti-inflammatory gels are sometimes helpful with neuropathic pain.

Previously, NMDA (N-methyl-D-aspartate) receptor antagonists such as ketamine and amantidine were infrequently used by infusion to treat neuropathic pain. Unfortunately, their pharmacological effects are short-lived and treatments need to be repeated. They also need to be given as an infusion in hospital with monitoring. This has made them unsuitable for the long-term management of chronic pain.

Opioids can be helpful in the management of neuropathic pain. If felt appropriate, patients could undergo an opioid trial. There should be an agreed plan between the patient's GP and the pain clinic as to prescribing responsibility and a single prescriber is recommended, the patient should be followed up regularly, the dose should not be escalated to excessive levels. If opioids do not improve the pain, then they should be stopped, even if no alternative is available. The long-term side effects of opioids are not fully understood, and we know that tolerance and dependence can develop. High doses can cause significant harm including death.

What injection treatments are used in neuropathic pain?

Local infiltration with steroid and local anaesthetic can be performed either into joints or soft tissue. Medial branch blocks and subsequent radiofrequency lesioning with under X-ray guidance is a common pain clinic procedure for back pain. These medial branch nerves supply sensation to the facet joints.

Lumbar epidural and caudal with steroid and local anaesthetic can be performed in the pain clinic for radicular leg pain.

Nerve root injection can be performed at any level of the spine for nerve root pain in a single dermatome. This type of pain can result from nerve root compression or following back surgery. Nerve root blocks can be performed diagnostically or therapeutically.

Soft tissue injections are performed for painful scar tissue, and into painful muscular trigger points.

Sympathetic blocks such as stellate ganglion block are used to moderate pain in the face and upper limb. Lumbar sympathetic block is used for sympathetically mediated pain in the lower limbs and is most useful for ischaemic leg pain, especially rest pain. Coeliac plexus block is most often indicated for the relief of pain from pancreatic carcinoma, but it can be used in managing pain from malignant disease of other viscera such as liver, stomach, spleen, kidney and colon.

What neuromodulation treatments do you know for chronic pain?

Transcutaneous electrical nerve stimulation or TENS is thought to work on the basis of the 'gate theory' of pain. TENS aims to stimulate the large myelinated A- β sensory fibres to block input from unmyelinated C-fibres. Pre-synaptic inhibition of C-fibres occurs with release of inhibitory neurotransmitters (such as GABA and glycine). Stimulation of the A- β fibres may also activate descending inhibitory pathways within the central nervous system to effect pain relief.

Other neuromodulatory devices are spinal cord stimulators, which also work via stimulation of A- β -fibres, but this time the large concentration of A- β fibres in the

dorsal columns are targeted, which is thought to cause inhibition of C-fibres and produce pain relief.

How is physical therapy important in chronic pain conditions?

Physiotherapy is important in chronic pain conditions, aiming to restore the function and mobility of damaged muscles and joints. Physiotherapists are an important part of the multidisciplinary pain clinic staff. Part of their role is to educate the patient in the active participation in managing their own pain.

How can the psychologist help in pain management?

Remembering that the definition of pain is a 'sensory and emotional response', it is not uncommon for people with chronic pain to suffer so much that they are unable to function, with breakdown of relationships and loss of their job. The psychological effects of chronic pain are negative thinking, stress, disability, loss of control and impaired mental and physical performance. Psychologists are involved in pain management programmes which are effective in improving quality of life. The aim in the pain management programme is to bring about an understanding of pain and alteration of beliefs about pain, using cognitive behavioural therapy. The patient learns coping skills and the aim is to reduce stress and disability caused by chronic pain.

CRPS

What is complex regional pain syndrome (CRPS)?

Don't be caught out if the examiners ask you what does the abbreviation CRPS stand for? It is NOT 'chronic' regional pain syndrome but 'complex' regional pain syndrome.

CRPS is a chronic pain condition, usually restricted to a particular region of the body. The primary symptom is continuous, severe pain which is out of proportion to the severity of the injury.

There are two types of CRPS:

- Type 1 was formerly known as reflex sympathetic dystrophy and occurs after a minor fracture or injury to a limb without direct injury to a nerve.
- Type 2 was formerly known as causalgia and develops after injury to a major peripheral nerve.

What are the clinical characteristics of CRPS?

There are four categories of signs and symptoms associated with CRPS. A diagnosis requires one symptom in three of the four categories and one sign in two or more categories, plus continuous disproportionate pain and no other diagnosis better explaining the signs and symptoms.

The categories are:

- Sensory with allodynia and/or hyperalgesia
- Vasomotor with changes in colour and/or temperature of the affected area
- Sudomotor/oedema with associated autonomic abnormalities such as hyper- or hypohidrosis and swelling

- Motor/tropic with abnormal nail/hair growth, fibrosis, osteoporosis and thin, glossy skin. Motor abnormalities such as weakness, dystonia, tremor, decreased range of motion.

What treatments do you know for CRPS?

Treatments are limited, but can be pharmacological, interventional, physical therapy or neurostimulation. A multidisciplinary approach is important and treatment should start immediately after diagnosis.

Pharmacological treatments depend on which mechanism is prominent and may include:

For inflammatory mechanism anti-inflammatory and/or immunomodulating drugs including steroids may be considered.

For pain and sensory disorders analgesics following the WHO analgesic ladder are tried; there is seldom sustained or significant benefit from these. Tricyclic antidepressants, the anticonvulsants such as gabapentin and pregabalin are also used as is topical capsaicin.

Vasomotor symptoms may be treated with vasodilator and motor disorders may warrant a trial of antispasmodic agents or muscle relaxants.

Interventional treatments used for CRPS include the sympathetic blocks such as stellate ganglion block and lumbar sympathectomy. Evidence for these is lacking.

Physical therapy is particularly important in the first few weeks of the diagnosis and patients must be encouraged to move the affected limb.

Psychological intervention is important as patients are often in significant distress.

Neurostimulatory treatments include TENS and spinal cord stimulation.

Trigeminal Neuralgia

What are the clinical features of trigeminal neuralgia?

The International Association for the Study of Pain (IASP) defines trigeminal neuralgia as 'a unilateral painful disorder that is characterised by brief, electric shock like pains, is abrupt in onset and termination, and is limited to the distribution of one or more divisions of the trigeminal nerve'. The pain can be bilateral in multiple sclerosis.

The pain can originate in the gasserian ganglion or the peripheral branches of the trigeminal nerve. Pain is typically provoked by innocuous stimuli such as light touch, eating, washing, talking or air currents on the face. It is more common in patients with multiple sclerosis and in patients over 50-years of age, but can sometime occur in young adults.

How is trigeminal neuralgia diagnosed?

It is often based on a clinical history. It is typified by paroxysmal attacks of facial pain in the distribution of the trigeminal nerve. It is often precipitated by trigger areas or factors, with pain being intense/sharp/superficial/stabbing or lancinating in nature. Investigation usually involves MRI looking for aberrant vessels and possible secondary cases.

What is the cause of trigeminal neuralgia?

The exact cause is not known. The majority of cases are thought to be due to demyelination around the root entry zone. This demyelination can be secondary to vascular compression near the pons, arterio-venous malformations, multiple sclerosis or tumours. An aberrant microvascular loop may be identified on MRI pressing on the nerve.

How can trigeminal neuralgia be managed?

Episodes of pain can initially be treated pharmacologically but if the patient fails to respond, then there are surgical options. Carbamazepine and oxcarbazepine are the first-choice drugs for trigeminal neuralgia. They both modulate voltage-gated sodium channels in neuronal tissue.

Gabapentin, pregabalin, amitriptyline and botulinum toxin B can also be used either alone or as additional therapy if symptoms persist. The muscle relaxant, baclofen is often used with other agents like carbamazepine or gabapentin to enhance pain relief in managing trigeminal neuralgia. There may be some benefit from lamotrigine and phenytoin.

If the symptoms fail to be controlled or there are significant side effects from drug therapy then surgery is considered.

Surgical treatments include microvascular decompression of an aberrant blood vessel, ablation of the ganglion using radiofrequency lesioning, glycerol injection, or mechanical balloon compression. All the ablative treatments have the potential to cause 'anaesthesia' dolorosa which is pain in an area of numbness.

Post-Herpetic Neuralgia

What are the clinical features of post-herpetic neuralgia?

Post-herpetic neuralgia is development of chronic pain 3 months after the resolution of an acute herpes zoster infection (shingles) infection. The commonest areas for shingles are the thoracic dermatomes and the ophthalmic division of the trigeminal nerve. The pain is commonly described as a constant, burning, throbbing, tingling, itching sensation. Symptoms included paraesthesia, sensory loss, and lancinating pain, often triggered by light touch or cold air. Signs include allodynia and hyperalgesia in the affected dermatomes. Post-herpetic neuralgia occurs in about 20% of all patients with herpes zoster.

Risk factors include:

- More intense acute pain
- Severe rash
- Prodrome of dermatology pain prior to rash development
- Older age
- Female sex.

Tell me about the pathophysiology of post-herpetic neuralgia.

Following a varicella zoster (chicken pox infection), the virus settles in the dorsal horns of the sensory ganglia and remains dormant. That is until reactivation of the virus produces inflammation in the peripheral nerve and neural destruction. This then causes pain in the distribution of that nerve. Reactivation of the virus is thought to be caused by immunosuppression, which is more likely with increasing age. Chronic pain is thought to arise due to the loss of nociceptive afferent neurones and A- β nerve fibre demyelination.

How is post-herpetic neuralgia managed?

Prevention is becoming a focus including varicella zoster vaccination and antiviral medications for acute episodes of shingles.

Treatments can be divided into three categories:

- Oral drugs
- Topical treatments
- Specific interventions or injections.

Oral drugs include antidepressants such as amitriptyline; the anticonvulsants gabapentin and pregabalin; conventional analgesics such as tramadol and opioids (which remain controversial).

Topical treatments include the use of capsaicin cream, non-steroidal anti-inflammatory creams and lidocaine patches.

Interventional treatments include TENS, sympathetic block of the stellate ganglion and epidural steroid and local anaesthetic at the level of the affected dorsal root ganglion. Evidence for these is limited.

Further Reading

Bharwani KD, Dirckx M, Huygen FJPM, Complex regional pain syndrome: Diagnosis and treatment, *BJA Education*. 2017; 17(8): 262–268.

Callin S, Bennett MI. Assessment of neuropathic pain. *Continuing Education Anaesthesia, Critical Care and Pain*. 2008; 8(6): 210–213.

Feller L, et al. Postherpetic neuralgia and trigeminal neuralgia. *Pain Research and Treatment*. 2017.

Gupta R, Smith PF. Post-herpetic neuralgia. *Continuing Education in Anaesthesia Critical Care and Pain*. 2012; 12(4).

Jones I, Johnson MI. Transcutaneous electrical nerve stimulation, *Continuing Education in Anaesthesia Critical Care & Pain*. 2009; 9(4).

Moore DM, McCrory C. Spinal cord stimulation. *BJA Education*. 2016; 16(8): 258–263.

Faculty of Pain Medicine. *Opioids Aware Resource*. www.fpm.ac.uk/opioids-aware-sitemap.

Vasappa CK, Kapur S, Krovvidi H. Trigeminal neuralgia. *BJA Education*. 2016; 16(10): 353–356.

Yao AL, Barad M. Diagnosis and management of chronic facial pain, *BJA Education*. 2020; 20(4): 120–125.

4.3.3 Low Back Pain – Murli Krishna and Kat Ng

How can we classify low back pain?

Low back pain can be classified on the basis of duration of symptoms as acute and chronic. It is defined as acute when symptoms have persisted for less than 6 weeks and chronic when symptoms last for longer than 3 months.

It can also be classified as ‘specific’ and ‘nonspecific’. Specific low back pain is caused by specific pathophysiological mechanisms such as herniated disc, infection, tumour, osteoporosis, inflammation or fracture. Nonspecific low back pain refers to those cases where no specific cause is found. Approximately 90% of all low back patients have nonspecific low back pain.

What are the causes of low back pain?

Try to classify by thinking in terms of causes i.e. . . . structural or inflammatory.

Table 4.3.3.1 Causes of low back pain

Structural	Spondylosis or spondylolisthesis Prolapsed intervertebral disc Facet joint arthritis Spinal stenosis
Neurogenic	Prolapsed disc Spinal stenosis Failed back surgery syndrome (arachnoiditis, epidural adhesions, recurrent disc herniations)
Inflammatory	Spondyloarthropathies Sacroiliitis
Neoplasm	Primary or secondary (metastatic)
Infection	Osteomyelitis Discitis Abscess
Metabolic	Osteoporosis Paget's disease Vitamin D deficiency Hyperparathyroidism
Referred pain	From visceral structures (pancreas, bowel), aorta, hip, retroperitoneal structures (kidneys)
Other	Somatoform disorders Fibromyalgia

How do you evaluate patients with low back pain?

Think in terms of aims of evaluation and targeted assessment/examination rather than head to toe examination (Table 4.3.3.1)

The aims of evaluation include:

- Identification of red flags (serious pathology)
- Identification of yellow flags (signs for chronicity)
- Identification of source of pain.

Assessment should include focussed history including nature of onset, duration, location of pain, radiation, neurological symptoms including bowel and bladder function, exacerbating factors, relieving factors. Ask specifically about rest pain and night pain as well as any history of trauma. Past medical history is very important especially history of malignancy, steroids, immunosuppression and intravenous drug abuse.

Examination should assess gait, posture, range of movements, tenderness on palpation and SLR test. Full neurological examination including tone, power, reflexes and sensory examination should be undertaken. Examination of other systems may be necessary in case of referred pain.

Investigations including blood tests, plain X-rays, MRI scans and bone scans as dictated by history, examination and findings.

Mnemonic worth remembering: OPQRST

Onset (spontaneous or triggered)

Pattern (location and timing)

Quality of pain

Relieving & exacerbating factors

Severity and Systemic Symptoms

Treatments

What are red flags and yellow flags?

Red flags suggest serious underlying pathology or nerve root pathology (Table 4.3.3.2).

Think in terms of red flag conditions, rather than symptoms. It is easier to remember and looks more impressive. This is most likely to feature in SAQs and possibly MCQs

Yellow flags are psychosocial barriers to recovery and indicate long-term chronicity and disability. They include:

- Belief that pain is damaging and activity is harmful
- Fear and avoidance behaviour
- Extremely high levels of pain
- Catastrophisation
- Low levels of activity
- Low mood, depression, social withdrawal
- Sickness behaviour (like extended rest)
- Tendency for seeking passive treatments
- Overprotective family or lack of support
- Problems at work, poor job satisfaction.

Table 4.3.3.2 Red flag conditions

Red flag condition	Symptoms
Cauda equina syndrome	Motor weakness, numbness, progressive neurological deficit Sphincter disturbance (urinary retention, bowel or bladder incontinence) Saddle anaesthesia
Tumour or infection	Age > 50-years or < 17-years-old Severe pain at rest, night time pain Acute localised bone pain Unexplained weight loss Fever, night sweats Immunosuppression, HIV, IV drug use
Fracture	Significant trauma Osteoporosis
Acute abdominal aneurysm	Pulsatile abdominal mass

Are you aware of any other flags?

Blue flags refer to conditions in the workplace that may inhibit recovery. These include high work demands, poor relationship with colleagues, low degree of control, monotony and lack of job satisfaction.

Black flags refer to organisational obstacles to returning to work. These include social benefits, sickness policies and compensation claims.

Orange flags include serious mental health issues in conjunction with pain. Examples include very high levels of distress, major personality disorders, posttraumatic stress disorder, substance abuse and clinical depression.

What is straight leg raise (SLR) test?

With the patient lying supine, the affected leg is raised with the knee fully extended. A positive test reproduces patient's leg pain (radicular pain) at between 30 to 70 degrees of elevation.

SLR tests the mobility of dura mater and the dural sleeve of lower lumbar and sacral spinal nerves (L4 to S3). It has high sensitivity for radiculopathy but low specificity.

What are signs of nerve root irritation?

- Leg pain greater than back pain
- Radiation into foot or lower leg
- Numbness and paraesthesias in dermatomal distribution
- Diminished leg reflexes
- Positive straight leg raising test (L4–S1 nerve roots)
- Positive femoral stretch test (L2–L4 nerve roots)
- Leg pain exacerbated by coughing, sneezing, or Valsalva manoeuvre.

What is cauda equina syndrome? How does it present and how should it be managed?

This is an important topic and could most likely feature in SAQs.

Cauda equina syndrome may result from any lesion that compresses the cauda equina nerve roots. This causes dysfunction of sacral and lumbar nerve roots in the vertebral canal. The syndrome is characterised by bladder (retention or incontinence), bowel or sexual dysfunction and perianal or saddle anaesthesia. Other clinical features include:

- Back pain
- Lower limb weakness
- Sensory changes in lower limbs
- Loss of (or sluggish) reflexes in lower limbs
- Unilateral or bilateral symptoms.

Patients can present either acutely as their first symptom of lumbar disc herniation, or after a long history of chronic back pain. It can also present insidiously with slow progression to numbness and urinary symptoms.

Features on examination include:

- Loss of perianal sensation
- Loss of anal tone
- Loss or diminution of lower limb reflexes

- Loss or diminution of bulbocavernosus reflex
- Motor weakness in lower limbs.

Describe your investigation and management of cauda equina syndrome.

MRI is the investigation of choice for confirming cauda equina syndrome. Clinical diagnosis has high false positive rates even in experienced hands. If MRI is unavailable, CT myelogram should be performed.

Surgical decompression is the treatment of choice for cauda equina syndrome. The timing of surgery remains controversial but retrospective analysis has shown that surgery within 24 hours is associated with a better outcome. The clinical outcome is poor in patients with retention and role of urgent surgery is less clear in these patients as shown by a recent meta-analysis.

What is the role of routine imaging in evaluation of low back pain?

There is no role for routine imaging in evaluation of low back pain. There is no evidence that routine plain radiography is associated with improvement in patient outcome in those with nonspecific low back pain. In addition, there is no evidence of causal relationship between radiographic findings and nonspecific low back pain. The amount of radiation from lumbar X-ray is also of concern (more than daily chest X-ray for a year). Similarly, up to 30% adults without low back pain have evidence of protruded disc on MRI scan.

Plain radiographs are recommended for initial evaluation of vertebral compression fracture in selected high-risk patients.

MRI scans or CT scans are recommended in patients with red flags (progressive neurological deficits, cauda equina syndrome, vertebral infection, cancer). Scans are also recommended if surgery or invasive treatment is planned.

What are the risk factors for chronicity in low back pain?

Always try to classify if possible.

Risk factors for chronicity can be divided in to three groups – individual factors, psychosocial factors and occupational factors (Table 4.3.3.3).

Table 4.3.3.3 Risk factors for chronicity

Individual factors	Obesity
	Low education level
	High levels of pain and disability
Psychosocial factors	Distress
	Depressive mood
	Somatisation
Occupational factors	Job dissatisfaction
	Heavy lifting
	Unavailability of light duties on return to work

What treatment options are available for low back pain?

Try to answer this question by dividing treatments in to specific groups (Non-pharmacological treatments, pharmacological treatments, interventional therapies and surgery).

1. Non-pharmacological treatments:

- Patient education – by providing evidence based information on low back pain, expected course, advise to remain active and effective self-care options (moderate quality evidence)
- Intensive multidisciplinary rehabilitation – pain management programmes, back pack programmes (moderate evidence)
- Exercise therapy
- Acupuncture
- Cognitive and behavioural therapy
- TENS machine (no proven benefit).

2. Pharmacological treatments:

- Oral analgesics: Paracetamol, NSAIDs, tramadol and strong opioids (controversial)
- Muscle relaxants: Benzodiazepines, indicated for short duration in acute back pain if significant muscle spasm present
- Antidepressants: Tricyclics (amitriptyline, nortriptyline) in low doses, more effective than placebo. SSRI are not particularly effective. SNRI (duloxetine, venlafaxine) have not been formally evaluated
- Gabapentanoids: gabapentin, pregabalin – for back pain associated with radiculopathy; however, use should be considered with caution due to risk of harm
- Systemic corticosteroids are not recommended.

3. Interventional treatments (Cochrane review 2008 concludes that there is insufficient evidence to support injection therapy in chronic low back pain but specific sub-group of patients may respond to specific injections).

- Epidural injection of steroids: short term benefit in radicular pain, no long-term improvement in pain or function
- Facet joint injections: Evidence equivocal (ASA practice guidelines 2010)
- Radiofrequency denervation of facet joints: better pain relief than sham interventions
- Spinal cord stimulators: for neuropathic leg pain (NICE approved).

4. Surgery – in selected cases (less than 1% cases)

- Discectomy/ microdiscectomy
- Spinal fusion
- Spinal decompression.

Further Reading

Low back pain and sciatica in over 16s:
Assessment and management. *NICE*

guideline. [NG59] last updated Dec 2020;
available from: www.nice.org.uk/guidance/ng59.

Practice Guidelines for Chronic Pain Management. *Anesthesiology*. 2010; 112 (4): 810–833.

Staal JB, de Bie R, de Vet HCW, Hildebrandt J, Nelemans P. Injection therapy for subacute

and chronic low-back pain. *Cochrane Database of Systematic Reviews*. 2008; (3). Art. No.: CD001824.

4.3.4 Assessment of Acute and Chronic Pain – Shelley Barnes, Sarah Love-Jone and Santhosh Gopalakrishnan

This topic can be asked as a part of the clinical sciences SOE. This is a tricky question, but a structured answer can get you through.

What is the definition of acute pain?

The IASP (International Association for the Study of Pain) definition of acute pain is ‘An unpleasant sensory and emotional experience associated with actual or potential tissue damage and expressed in terms of such damage’.

What can you tell me about pain assessment?

Pain is most commonly measured by pain measurement scales, which use aspects of patient reporting that can yield reproducible data. These should be easy to use and interpret over a wide variety of disease states and cultures.

Outline the different methods of measuring pain that you know of.

A structured approach is vital to ensure that you do not forget any of the key points. It may be worth making an initial broad classification.

Pain is measured mainly using pain measurement scales. They can be broadly classified into:

Simple pain scales used to assess acute pain and multidimensional pain scales used to assess chronic pain.

What are simple pain scales and how do you interpret them?

Simple pain scales are widely used to measure acute pain intensity due to the ease of use.

The most commonly used scales are:

- Verbal rating scale
- Numerical rating scale
- Visual analogue scale.

Tell me about the verbal rating scale.

The verbal rating scale (VRS) has a number of adjectives to describe pain. These are no pain, mild pain, moderate pain, severe pain, very severe pain and worst possible pain. The verbal rating scale is commonly used in postoperative recovery rooms because it is simple to use. It is, however, insensitive to small changes in the patient’s pain intensity.

Describe the numerical rating scale (NRS).

The numerical rating scale (NRS) takes two extremes of pain (no pain and worst possible pain) with numbers across the scale from zero to ten, making an eleven-point scale. The patient is asked to translate their pain severity into a number where zero is no pain and ten is the worst pain they can imagine. The numerical rating scale is not very sensitive in measuring small changes in pain intensity, but is simple to use and can be translated into different languages.

What is the visual analogue scale (VAS)?

The visual analogue scale (VAS) is a simple pain scale, often used in research. The patient is shown a 10 cm line with the anchor words 'no pain' written at one end and 'worst pain possible' written at the other end. The patient is asked to put a cross on the line that best describes their pain. The distance from the 'no pain' end is then measured in centimetres to give the pain score. This continuous aspect of the scale differentiates it from discrete scales. The visual analogue scale is sensitive to small changes in patient's pain.

What are multidimensional pain scales?

Simple pain scales do not separate the many factors associated with chronic pain. Chronic pain is of longer duration, is difficult to manage and often of unknown origin. Psychosocial factors play a large part in the development of chronic pain and the patient and their immediate family will often be distressed and fearful about its impact on their life, work and finances. The multidimensional pain scales analyse chronic pain by specific enquiry and can establish problems that do not just include pain intensity. The multidimensional pain scales include the McGill Pain Questionnaire, the Brief Pain Inventory, the Neuropathic Pain Scale and the Leeds Assessment of Neuropathic Symptoms and Signs.

Can you briefly describe the McGill Pain Questionnaire?

The McGill Pain Questionnaire is a three-part assessment tool.

Part 1 is an anatomical drawing on which the patient marks the location of their pain.

Part 2 is a verbal descriptor scale

Part 3 is a verbal descriptor inventory of over 70 adjectives.

These word descriptors are sensory, affective, evaluative and miscellaneous and are used by patients to specify subjective pain experience. The McGill Pain Questionnaire provides quantitative information that can be treated statistically, and is sufficiently sensitive to detect differences amongst different methods to relieve pain. It also has a shorter version, with only 15 words, which is more commonly used nowadays.

Outline the short form McGill Pain Questionnaire (SF-MPQ).

The main component of the SF-MPQ consists of 15 descriptors (11 sensory; 4 affective) which are rated on an intensity scale as 0 none, 1 mild, 2 moderate or 3 severe. Three pain scores are derived from the sum of the intensity rank values of the words chosen for sensory, affective and total descriptors. The SF-MPQ also includes the Present Pain Intensity (PPI) index of the standard MPQ and a visual analogue scale (VAS).

What is meant by a Brief Pain Inventory?

The Brief Pain Inventory assesses pain intensity at its best and worst and at the time of the test. It also assesses the percentage relief from medications and treatments for pain and the duration of relief. There are specific questions about patient's beliefs regarding their pain and questions about how their pain interferes with their daily activities such as work, relationships, rest and sleep.

Describe the Neuropathic Pain Scale.

The neuropathic pain scale specifically assesses neuropathic pain. It has eight descriptors of pain quality. There are eight descriptors:

- Sharp pain
- Hot
- Dull pain
- Cold
- Skin sensitivity
- Itching
- Surface pain
- Deep pain.

It also measures the unpleasantness of pain on a scale of zero to ten.

What is the Leeds Assessment of Neuropathic Symptoms and Signs (LANSS)?

The LANSS system identifies patients in whom neuropathic pain is the dominating part of their pain. It is based on five questions and two simple bedside clinical tests to assess sensory dysfunction (presence of allodynia and pinprick tests). It takes approximately 5 minutes to complete.

What are the adjunctive measurements of pain?

In chronic pain, psychological distress and social function are important assessments. These adjunctive measurements can be divided into:

- Assessment of mood
- Quality of life

The assessment of mood enables clinicians to tailor individual treatments for each patient and evaluative questionnaires are often used. The commonly used ones are the Beck anxiety and depression inventories, hospital anxiety and depression score and Zung self-rated depression score. Quality of life assessments aim to evaluate the impact of the disease on the patient's life.

How do you assess pain in children?

It is very difficult to assess pain in children. Scales for children must be easy to use and be reproducible.

Up to 3 years of age?

Neonates can feel pain contrary to old beliefs and theories. Effective pain scoring systems must rely on behavioural variables such as facial expression and crying or variables like cardiovascular parameters. The commonly used scales are the objective pain scale and the clinical scoring system. These use a combination of physical, physiological and nurse perception variables.

3–7 years of age?

Most children above 3 years of age can report pain and its intensity effectively. Thus, a verbal rating scale or the Wong-Baker Faces scale can be used. The Faces scale is a series of cartoon faces that range from smiling to crying. Visual analogue scales can also be used and better results can be obtained by using the colour graduation system and making the line vertical.

More than 7 years of age?

Conventional visual analogue scales can be used.

How can pain be assessed in the cognitively impaired patient?

It can be assessed using the behavioural pain assessment scale, known as the FLACC score.

Pain is scored from 0–10 and is based on:

- Face Score
- Legs/restlessness Score
- Consolability Score
- Activity/muscle Tone Score
- Cry/vocalisation Score

Zero = no evidence of pain. Mild pain = 1–3. Moderate pain = 4–5. Severe uncontrolled pain is ≥ 6 .

Further Reading

Bendinger T, Plunkett N. Measurement in pain medicine. *BJA Education*. 2016; 16(9): 310–315.

Breivik H, Borchgrevink PC, Allen SM, Rosseland LA, Romundstad L, Breivik Hals EK, Kvarstein G, Stubhaug A. Assessment

of pain. *British Journal of Anaesthesia*. 2008; 101(1): 17–24.

Callin S, Bennett MI. Assessment of neuropathic pain. *Continuing Education in Anaesthesia, Critical Care and Pain*. 2008; 8 (6): 210–213.

4.3.5 Analgesic Techniques – Gary Thomas

Can you describe what is meant by the ‘analgesic ladder’?

The World Health Organisation (WHO) originally described a three-step approach to the administration of analgesics for cancer pain. If the patient’s pain does not respond

initially to weak peripherally acting non-opioid analgesics, the first rung of the ladder, then there are two further steps that allow the escalation of therapy. It has been adapted to encompass the management of acute pain by The World Federation of Societies of Anaesthesiologists (WFSA).

Pain is assessed and can be categorised as mild, moderate or severe. Treatment begins according to which level of pain the patient is experiencing; therefore, the treatment of a patient in severe pain need not start at the first step. If the pain becomes more severe or the analgesia is ineffective, then the patient moves up one step of the ladder to the next level of pain severity and is treated accordingly.

Can you describe the steps of the original ladder?

The three steps of the ladder ascend from non-opioids through weak opioids to strong opioids, according to the severity of the pain. For mild pain, non-opioid analgesics such as paracetamol and NSAIDs should be prescribed regularly. For moderate pain, weak opioids such as codeine are added, and for severe pain strong opioids such as morphine or fentanyl are substituted for the weak opioids.

In addition to analgesia, care must be taken to assess the patient for nausea and vomiting and sedation and respiratory depression.

What are the advantages and disadvantages of the analgesic ladder?

The advantages are:

- It is simple to use
- It is useful for a variety of clinical situations
- It employs commonly used drugs
- It emphasises a multimodal approach to pain relief.

The disadvantages are:

- It is not easily applicable to different forms of chronic pain
- It may be inappropriate for some patients to take oral drugs
- The use of regional anaesthesia does not fit within the original ladder structure.

An ASA I, 45-year-old woman has presented for an elective total abdominal hysterectomy. How would you manage her postoperative pain?

This answer should not involve too much detail – the examiner will have further questions ready to tease out the finer points.

Abdominal hysterectomy usually involves a transverse incision below the umbilicus and is associated with moderate postoperative pain. My practice is to use a combination of oral and intravenous analgesic drugs and peripheral nerve blocks that are performed following induction of anaesthesia.

Bilateral inguinal nerve blocks can be performed but I prefer to carry out a bilateral transversus abdominis plane (TAP) block. The latter can provide between 12 and 24 hours of pain relief.

The oral analgesics I prescribe regularly are paracetamol and diclofenac, providing there are no contraindications to their use.

I also prescribe intravenous morphine via a PCA device, though it could be given as required using the intramuscularly route. A potential disadvantage of morphine in gynaecological surgery is a high rate of nausea and vomiting. Antiemetic should also be prescribed to counteract this.

The epidural route is not commonly used but may be indicated in certain patients, for example when trying to avoid morphine in patients with obstructive sleep apnoea.

You have mentioned a Transversus Abdominis Plane (TAP) block. Can you describe the anatomy relevant to this block and how you would perform it?

TAP blocks have become increasingly popular recently and so a good knowledge of the relevant anatomy would be expected.

The anterior rami of spinal nerves T7 to L1 innervate the anterolateral abdominal wall. Branches from the anterior rami include;

- The intercostal nerves (T7–T11),
- The subcostal nerve (T12), and
- The iliohypogastric and ilioinguinal nerves (L1).

At points in their course these nerves run in the plane between the transversus abdominis and internal oblique muscles before ending as cutaneous branches supplying the skin of the front of the abdomen.

A single injection will most likely block T10 to L1 and for an incision across the midline, as you would expect for an abdominal hysterectomy, bilateral blocks are required. The block can be performed using a landmark technique or using ultrasound. Classically, the point of entry is the 'triangle of Petit' which is situated between the lower costal margin and iliac crest. When using the landmark technique, the endpoint, a double pop is felt as a blunt needle passes through the external oblique and internal oblique muscles. Use of ultrasound requires the probe to be placed in the transverse plane to the lateral abdominal wall in the midaxillary line.

Success depends on a good spread of local anaesthetic and so 25–30 ml of local anaesthetic on each side should be used, adhering to the maximum safe dose.

What are the advantages and disadvantages of Patient Controlled Analgesia (PCA)?

The advantages of PCA are:

- Provide an on-demand system minimising the delay between the experience of pain and receiving analgesia
- Consistent drug plasma concentration when compared to intramuscular techniques and therefore better level of analgesia
- Reduction of nurses' workload
- Allows the patient to be in control which may contribute to a placebo effect and possibly better outcome
- Daily analgesic dose or number of demands can be used as an objective measure to monitor progress

The disadvantages include:

- Need for expensive equipment
- Risk of patient harm from incorrect programming or equipment malfunction
- Cannot be operated by some patients, i.e. physical or cognitive impairment.

What other routes for the administration of analgesia do you know?

Analgesic drugs can also be given intranasally, sublingually and transdermally.

Tell me about transdermal delivery systems.

The advantage of transdermal route for drug delivery is that it avoids first pass metabolism and large variations in plasma drug concentration. Drugs suitable for transdermal administration must be able to penetrate the stratum corneum of the skin, which is highly lipophilic. To this end drugs must have a low molecular weight and high lipid solubility.

There are two types of transdermal patch available: the reservoir, or membrane-controlled system, and the matrix system. In a reservoir patch the drug is held in a gel or solution and a release membrane controls the rate of delivery of the drug to the skin. The matrix patch holds the drug as part of a polymer matrix. The matrix is applied directly to the skin.

Which analgesic drugs can be delivered via a transdermal patch and when are they indicated?

The most common analgesic drugs are buprenorphine and fentanyl. These opioid patches are not suitable for acute pain management because of the delay between patch application and the development of a desired minimum effective concentration. They are frequently prescribed to patients with chronic or cancer pain.

Tell me what you know about fentanyl patches.

Fentanyl patches are more commonly of the matrix type. The patches deliver fentanyl at a constant rate and come in different strengths: 25 µg per hour up to 100 µg per hour. A steady state serum concentration is achieved after 24 hours and will continue as long as the patch is renewed. Each patch lasts for 72 hours.

Recently developed iontophoretic fentanyl drug delivery devices enable the patient to activate the delivery of a small dose of ionised analgesic transdermally using an electric current. In contrast to the reservoir and matrix patches, these are licensed for the treatment of acute postoperative pain.

What are the advantages and disadvantages of opioid transdermal patches?

Because the constant delivery of the drug avoids the peaks and troughs of intermittent dosing the side effect profile of opioids, such as sedation, nausea and vomiting and respiratory depression, is reduced. Patient compliance is also improved because of their convenience.

A problem common to all transdermal patches is skin irritation which can be solved by removing the patch. Respiratory depression and sedation can still occur and care must be taken when using other sedatives. Removing the patch will not immediately stop these problems and so patients and their carers have to be well educated as to the signs and symptoms of impending side effects.

Drug abusers have been known to extract the fentanyl from patches to inject intravenously.

What pharmacological adjuncts are used to treat pain?

Most adjuncts are used to treat chronic pain syndromes, in particular neuropathic pain. This should be emphasised at the start. The answer to this question then lends itself to a simple classification.

Acute inflammatory pain is usually well managed with a combination of paracetamol, non-steroidal anti-inflammatory drugs and morphine. These analgesics are often less effective when the patient is experiencing neuropathic pain. Drugs not developed to treat acute pain have been shown to be effective in the management of neuropathic pain. These include tricyclic antidepressants, anticonvulsants, membrane stabilisers and other miscellaneous drugs.

Tricyclic antidepressants (TCAs) such as amitriptyline have been used in low dosages to treat neuropathic pain. They work by inhibiting the reuptake of neurotransmitters noradrenaline and serotonin (5-HT) and so potentiate the inhibitory pathways that occur in the dorsal horn of the spinal cord. The TCA mechanism of action may also involve sodium-channel blockade or N-methyl D-aspartate (NMDA) receptor antagonism.

Anticonvulsants are used to treat neuropathic pain; in particular, carbamazepine is the first line treatment for trigeminal neuralgia. Neuropathic pain is associated with ectopic discharge from sodium channels. Carbamazepine is also a sodium-channel blocker and this is thought to be why it can be effective. Gabapentin is also an anticonvulsant and is structurally related to gamma aminobutyric acid (GABA). Its site of action appears to be the $\alpha_2 \delta$ subunit of voltage-dependent calcium channels although its overall mechanism of action is not understood. It has been shown to be effective in the management of post-herpetic neuralgia and in particular diabetic neuropathic pain. Other anticonvulsants used in chronic pain syndromes include phenytoin, fosphenytoin and lamotrigine.

Membrane stabilisers such as lidocaine are used to treat chronic pain. Infusions of lidocaine are used to treat the pain associated with fibromyalgia syndrome and lidocaine patches are now licensed for use in the management of post-herpetic neuralgia. Its mechanism of action may again be through the suppression of ectopic discharges from sodium channels.

Miscellaneous drugs used in the treatment of chronic pain include:

- Baclofen – Inhibition of the release of the excitatory neurotransmitters, glutamate and aspartate
- Proglumide – Inhibition of cholecystokinin (CCK)
- Ketamine – NMDA receptor antagonist
- Clonidine/dexmedetomidine – Centrally acting alpha-2 adrenoreceptor antagonists
- Capsaicin – Reversible inhibition of substance P release from sensory nerve endings.

What non-pharmacological techniques can be used in the treatment of pain?

There are a number of non-pharmacological techniques that can be used to manage pain. They are rarely used as isolated interventions but are more often part of a multidisciplinary approach to pain management, particularly in the chronic pain setting.

Psychological intervention forms an important part of chronic pain management and can have a role in acute pain. Simple steps such as providing the patient with information about any forthcoming procedure has been shown to reduce pain scores and lengths of hospital stay. Relaxation training can be beneficial, as can hypnosis.

Cognitive behavioural therapy interventions focus on trying to alter targeted behaviours and utilise positive reinforcement of desired behaviours, pacing of behaviours and goal setting. It often occurs in a group setting and requires the patient to be an active participant in the process.

Transcutaneous electrical nerve stimulation, or TENS, is commonly used to help manage pain in the early stages of labour and is used to treat a variety of chronic musculoskeletal pains in chronic pain clinics. There are two theories proposed to explain its action. In the gate control mechanism, TENS stimulates large diameter A β fibres that inhibit the onward propagation of nociceptive signals via small diameter C-fibres within the dorsal horn of the spinal cord. The second theory is that TENS stimulates the release of endorphins that act on receptors within the central nervous system.

Acupuncture can be effective in the treatment of pain although its use and effectiveness remains controversial.

Environmentally Sustainable Anaesthesia

Rebecca Taylor-Smith and Paul Southall

What is the environmental impact of healthcare and why is it important?

Sustainable healthcare meets the needs of the present generation without compromising the ability of future generations to meet their own needs.

The NHS contributes approximately 5% of the UK carbon footprint. This is important because the climate crisis is a health crisis. Heatwaves, air pollution and increased infectious diseases threaten the health of our patients and widen health inequalities. Reducing our carbon emissions will help to save lives, reducing cases of asthma, cancer and heart disease. Particularly relevant to anaesthetists is the opportunity this offers to optimise patient health, reduce complications in the perioperative period and thus further reduce the carbon emissions of healthcare.

The NHS was the first national health system in the world to set a target for reaching 'Net Zero' carbon – that is to reach 'net zero' for the emissions we control directly (the NHS Carbon Footprint) by 2040 and for the emissions we can influence (our NHS Carbon Footprint Plus) by 2045.

Describe the highest areas of carbon emissions in anaesthetic practice.

Anaesthetic gases contribute 2% of NHS carbon emissions (Figure 5.1.1) and 42% of the carbon emissions of operating theatres. Volatile agents are greenhouse gases and each has a different global warming potential (GWP). Sevoflurane, isoflurane and desflurane have 130, 510 and 2540 times the GWP of carbon dioxide over a 100-year time horizon (GWP100), respectively.

Nitrous oxide (N_2O) is a greenhouse gas and accounts for around two thirds of carbon emissions from anaesthetic gases. Its GWP is 265 times that of CO_2 , it is an ozone depleter and it persists for 114 years in the atmosphere.

Medicines account for 25% of the NHS carbon footprint, with intravenous medicines generally having a higher carbon footprint than oral medicines due to sterilisation, packaging and transport emissions.

Operating theatres are one of the most resource-intensive locations in healthcare. Annually, each operating theatre generates 2.3 tonnes of solid waste and the equivalent of 188 tonnes of CO_2 . Areas we can tackle include reusable equipment, energy usage, waste management and efficient care pathways.

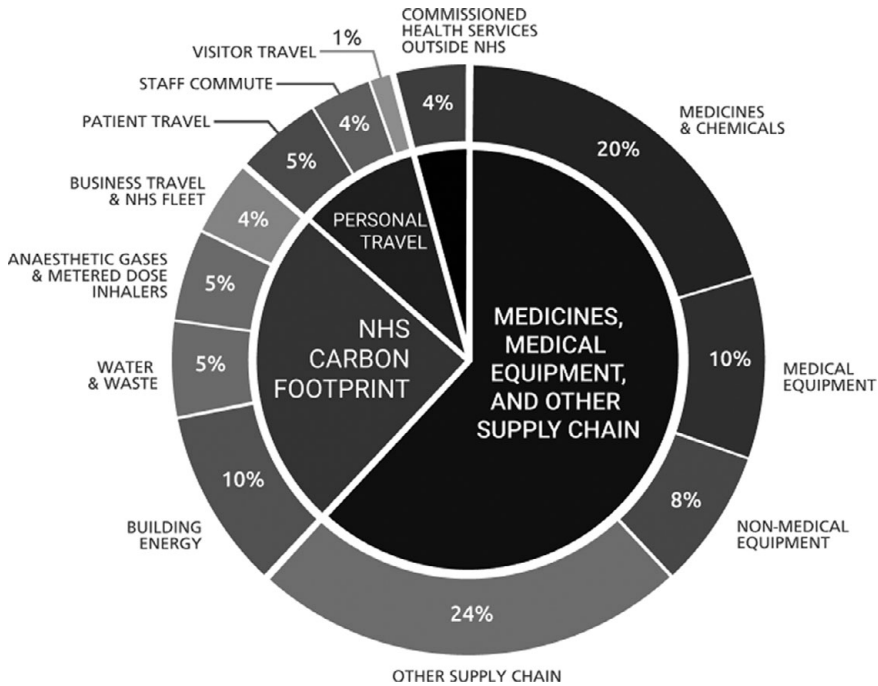


Figure 5.1.1 Sources of carbon emissions by proportion of NHS Carbon Footprint Plus. Source: Delivering a 'Net Zero' National Health Service, 2020

What strategies can be employed within anaesthetic practice that can reduce the carbon impact of anaesthesia, without impacting on clinical care?

To reduce the carbon impact of anaesthetic practice we can consider direct factors and indirect factors.

Direct factors are directly related to the induction and/or maintenance of anaesthesia and include choice of anaesthetic such as volatile, intravenous, regional or local anaesthesia. We can reduce volatile usage by using total intravenous anaesthesia (TIVA) or regional anaesthesia, selecting the volatile with the lowest GWP, utilising low flows and avoiding nitrous oxide where clinically safe to do so.

Indirect factors are those which can be impacted on by anaesthetic practice such as packaging and equipment, methods of patient warming and theatre energy usage, and resource use in the perioperative pathway. We can reduce emissions associated with these by opting for reusable equipment in some cases, reducing unnecessary glove usage, using conductive rather than convective warming, encouraging the switching off of scavenging systems (AGSS) overnight and reducing unnecessary duplication and variation in the perioperative pathway.

What methods can we use to reduce the environmental impact of volatile anaesthetics and nitrous oxide specifically?

There are several methods to reduce the environmental impact of a general anaesthetic. The simplest way to eliminate the impact of anaesthetic vapours and nitrous oxide

is to avoid their use entirely, favouring TIVA or regional anaesthesia, if clinically appropriate.

Where a volatile anaesthetic is required, selecting a volatile with the lowest GWP, such as sevoflurane; minimising gas flow rates (to less than 1 litre) and avoiding the use of nitrous oxide would lower the carbon emissions. Vapour capture technology is an emerging technology which could significantly lower the carbon emissions of a volatile anaesthetic. Processed EEG monitoring could be used to provide the minimum effective amount of anaesthetic.

In terms of nitrous oxide we can consider using alternative methods of delivering anaesthesia and analgesia. Alternative analgesics include methoxyflurane (which is sometimes used in the pre-hospital or A&E environment) or remifentanyl for labouring parturients. In the absence of an appropriate substitute, emerging technology for 'cracking' nitrous oxide into nitrogen and oxygen and its use is currently being evaluated and this might be best used in two areas of anaesthetic practice – obstetrics and paediatrics – where nitrous oxide is still currently widely used.

One of the greatest sources of emissions is wastage of nitrous oxide products from leaking pipes, expiring cylinders and theft. Wherever possible, nitrous oxide cylinder manifolds should be decommissioned as a priority, instead using smaller cylinders attached to the back of individual anaesthetic machines directly if required.

What environmental issues does TIVA present?

The overall impact of TIVA has been demonstrated to be several orders of magnitude lower than vapour-based anaesthesia, even considering the increased requirements for plastic consumables that TIVA requires. A full comparison of the reduced impact of TIVA requires a comprehensive lifecycle analysis (LCA) of all the components and drugs required – including the energy source that powers both the factories where the consumables and drugs are produced, as well as the theatre in which they are used.

However, there are other environmental issues associated with its use. Propofol is known to be a potent aquatic toxin, and is only degraded by very high temperatures. Propofol that is not disposed of correctly, such as in the clinical waste stream or poured down the sink, has a high chance of entering the watercourse, with potential deleterious effects on aquatic fauna and flora. There is not yet sufficient evidence to ascertain whether the metabolic products of propofol metabolism (such as propofol glucuronide) are also potentially harmful to aquatic life. This area of research needs to progress before robust comparisons between vapour-based anaesthesia and TIVA can be made.

Are there any other areas we can influence within operating theatres?

Appropriate waste management in theatres

There are significant financial and carbon costs between the different waste streams in the hospital setting. Where possible, waste that is obviously recyclable (such as paper drug information pamphlets) should be disposed of in dry mixed recycling. The majority of anaesthetic waste does not need to be disposed of in clinical waste streams, as it consists largely of packaging that has not come into direct contact with a patient, and should be disposed of either in dedicated recycling schemes (if available) or in 'black bag'

waste if not available. Utilising lower temperature incineration waste streams for non-infectious waste such as 'tiger bags' will also reduce environmental impact.

Increased use of reusable anaesthetic equipment

The carbon cost of reusable anaesthetic equipment (such as laryngoscopes) has been demonstrated to be several orders of magnitude lower than the equivalent single use item. Where possible, a shift towards reusable anaesthetic equipment should be heavily encouraged.

Reducing unnecessary energy usage in theatres

Out of hours, computers should be shut down, and where technically possible, AGSS systems and the anaesthetic machines themselves should be switched off when the theatre is not in use

How can anaesthetists impact the wider carbon impact of healthcare outside of the theatre environment?

Anaesthetists are regularly exposed to a broad range of other clinical specialities. This places us in a unique position to assist with improvements in clinical sustainability projects across secondary care. One of the greatest sources of emissions is wastage of nitrous oxide products from leaking pipes, expiring cylinders and theft. It is important for hospitals to reduce this as well as consider clinical solutions, and anaesthetists are key team members in the success of this work.

As perioperative physicians we can influence the perioperative patient pathway. Good quality care is low carbon care and engaging with prehabilitation and Getting it Right First Time (GIRFT) programmes tends to lead to improved health outcomes and lower healthcare resource use. Reducing unnecessary duplication through amalgamating pre-operative assessment visits and making use of digitally enabled care where appropriate are important goals factors to consider in reducing the carbon embedded in patient pathways.

What is the triple bottom line?

This is a term used in quality improvement. It recognises that environmental, social and financial resources are finite. By considering the triple bottom line we can deliver an excellent standard of care to patients at the minimal financial and environmental cost whilst adding positive social value. An example of this could include a prehabilitation programme that improves patient health and wellbeing (social value) and reduces post-operative complications resulting in a financial and environmental saving:

$$\text{Value} = \frac{\text{Outcomes from patients and populations}^1}{\text{Environmental} + \text{Social} + \text{Financial impacts} \text{ (the triple bottom line)}}$$

¹ The triple bottom line equation. From Sustainability in quality improvement: Redefining value. F Mortimer et al. Future Healthcare J 2018;5:88–93.

Additional Questions

How does volatile capture technology work?

These techniques utilise a canister which sits downstream of the exhaust pipe of the anaesthetic machine but upstream of the common outlet for the theatre anaesthetic gas scavenging system (AGSS). Typically, these canisters contain an activated substance (such as coconut husk) in pellet form, which will adsorb the anaesthetic vapours, similar to the theory behind the Cardiff aldasorber. However, unlike the Cardiff aldasorber, these canisters can be heated once full, releasing the vapours unchanged, which can then be redistilled, rebottled, and reused, potentially an infinite number of times. The main drawbacks to these systems are the cost to purchase the required equipment, and the ongoing costs associated with their continued use (one canister can hold approximately one bottle of vapour before it requires changing). The major advantage is that it requires no alteration in current anaesthetic practice. Research into its effectiveness is ongoing.

How does nitrous oxide cracking technology work?

The only version of such technology that is currently widely available destroys nitrous oxide by heating it to 400°C, which splits the nitrous oxide into oxygen and nitrogen. Such devices are known as ‘catalytic crackers’, and have been used extensively in Scandinavia for many years. These devices require only a power source, and no additional ventilation, and are available as portable devices or a larger central unit.

Portable devices would be used with a single anaesthetic machine or attached to the exhaust port of a demand valve such as those commonly used in maternity and A&E. Larger central units utilise the AGSS to deliver the waste nitrous oxide/mixed nitrous oxide to the device. Unlike anaesthetic vapour scavenging systems, these central devices sit further downstream where several AGSS outlets converge into a common exhaust.

Further Reading

NHS England. Delivering a ‘Net Zero’ National Health Service. 2022; www.england.nhs.uk/greenernhs/publication/delivering-a-net-zero-national-health-service/.

Chakera A, Fennell-Wells A, Allen C. Piped nitrous oxide waste reduction strategy. *Association of Anaesthetists*. 2021; <https://anaesthetists.org/Portals/0/PDFs/Environment/Nitrous%20waste%20methodology.pdf?ver=2021-04-26-115439-240>.

White SM, Shelton CL, Gelb AW, Lawson C, McGain F, Muret J, Sherman JD, representing the World Federation of Societies of Anaesthesiologists Global Working Group on Environmental Sustainability in Anaesthesia. Principles of environmentally-sustainable anaesthesia: A global consensus statement from the World Federation of Societies of Anaesthesiologists. *Anaesthesia*. 2022; 77 (2): 201–212.

Ethics and Consent

Ruth Doolin

How do you assess whether a patient has capacity to consent?

Capacity is a two-stage test. Firstly, the question of whether the person has an impairment of their brain – this could be long-standing result of illness or injury, or temporary, secondary to recent drug or alcohol use. Secondly, does the impairment affect the ability to make a specific decision. A patient should be able to demonstrate they have the ability to understand and retain the information, weigh up the options and communicate their decision.

What is meant by the Mental Capacity Act?

The Mental Capacity Act (MCA) of 2005 protects people in England and Wales unable to make some or all of their decisions for themselves. It says that a patient is always assumed to have capacity, unless it has been proven that capacity is lacking. We do not always have to agree with a patient's decision. The MCA protects the right to make an unwise decision. Wherever possible people should be supported to make their own decisions, this may be through assessing capacity at a different time in case their condition is fluctuant, exploring different types of communication e.g. written or digital.

When making a decision for someone lacking capacity, it must be in their best interests. Where treatment is being provided for someone without capacity, the least restrictive option should be given.

What is an independent mental capacity advocate?

An Independent Mental Capacity Advocate (IMCA) is instructed for a person lacking capacity to make a decision, without any other support. This could be in relation to serious medical treatment, or non-medical matters. The role is to represent the views of the patient as part of the process of working out which actions are in the patient's best interest.

What is an advance directive?

An advance directive allows someone over the age of 18 years with capacity, to make decisions about their future healthcare options when they may lack capacity to decline or consent to treatment. It should be in written form, signed and witnessed. An advance directive is a legal document that must be followed by healthcare professionals unless they have evidence or reason to believe a person's views or opinions have changed since it was written.

What is a lasting power of attorney?

A Lasting Power of Attorney (LPA) is a legal document allowing a patient to nominate another person to make decisions on their behalf. The decision of the LPA is as valid as the decision of the patient if they were able to make the decision themselves. A Lasting Power of Attorney can be nominated for different areas, namely, property and financial affairs LPA or health and welfare LPA. These can be appointed separately. To make decisions regarding healthcare, an LPA for health and welfare is required.

What are the different types of consent?

Consent can be implied or expressed. Implied consent is assumed by the actions of a patient, for example offering an arm for venepuncture. Expressed consent is usually written or oral and should be sought where a procedure carries any significant risk. Consent should be documented in the patient's notes.

What are the different types of consent form?

A consent form type 1 is used for a patient to consent to treatment or investigation. Consent form type 2 is used for a parent consenting on behalf of a child or young person. Consent form 3 allows patients to consent to treatment under local anaesthesia, or where consciousness is not impaired. Finally, a consent form 4 is used where a patient lacks capacity to consent for themselves.

At what age are children assumed to have to competence to consent?

According to law, all people over the age of 16 are assumed to be competent to give consent to medical procedures in England, Scotland, Wales and Northern Ireland.

Can a person under the age of 16 be competent to consent?

Yes. This is sometimes referred to as Gillick competence, based on a court ruling in 1983 when a mother challenged the Department of Health guidance allowing contraceptive prescriptions to girls under 16 without the knowledge of parents. The Gillick test allows children under the age of 16 to give consent to treatment, providing they have 'sufficient understanding and intelligence to fully understand what is involved in a proposed treatment, including its purpose, nature, likely effects, risks, chances of success and availability of other options'.

Whilst a patient under the age of 16 is able to consent to treatment if they are Gillick competent, they are not able to refuse the treatment if it is deemed to be in their best interests, until they are 18 years of age. In this situation the decision can be overruled by a person with parental responsibility or by a court; however, their view should be taken into account.

Who is able to consent on behalf of a child or young person lacking competence?

A person with parental responsibility is able to consent, provided the decision is in the best interests of the child.

What happens when the decision is not in the best interests of the child?

Lifesaving treatment may be given whilst a court order is sought. However, the least restrictive options should be explored.

If looking after a patient with a Do Not Attempt Resuscitation (DNAR) directive during the perioperative period, how would you approach this?

Where possible this should be reviewed preoperatively to decide if it may be appropriate to suspend or alter the order. Ideally this discussion should include the patient, alternatively the LPA to discuss specific treatments and eventualities. Outcomes of these discussions could include a decision for full resuscitation during the perioperative period, limited attempts at resuscitation with respect to particular treatments, e.g. cardiopulmonary resuscitation or defibrillation, or limited attempts at resuscitation with respect to the patient's goals e.g. to manage quickly reversible situations but not those which may have long-term sequelae e.g. treating a bradyarrhythmia associated with insufflation during laparoscopic surgery. These conversations and decisions should be documented in the patient's notes alongside a plan for re-initiation of the original DNAR.

When looking after a patient from the Jehova's Witness community undergoing elective major surgery, how would you approach management of blood products?

This plan should be discussed preoperatively involving the full multidisciplinary team, including the patient, surgeon, anaesthetist, and haematologist. Each patient should be given a thorough explanation of procedures related to blood transfusion that may be required, risks of declining and alternatives available. Any treatments the patient accepts or declines should be documented. Decisions should be made freely and without coercion from either medical or religious sides. Any pre-existing anaemia should be treated, and clotting deficiencies should be identified and corrected. It is recommended to check and replace iron, ferritin and folate.

Intraoperatively, the patient's preferences should be discussed as part of the team brief. Techniques to minimise blood loss should be used where possible, including cell salvage, controlled hypotension, minimising blood sampling, normothermia and elective use of tranexamic acid.

Postoperatively, a comprehensive written and verbal handover should be given to the postoperative care area, alongside the patient's wishes. The aim is to minimise ongoing blood loss, promoting haemostasis and correcting coagulation defects, optimising oxygen delivery and consumption.

How would you approach a patient from the Jehova's Witness community who presents requiring a blood transfusion, unable to communicate their wishes?

In emergencies, the team should treat the patient to the best of their ability. If there is an advance directive, this should be respected and followed provided there is no reason to

believe the patient's views may have changed in the intervening time. In the absence of an advanced directive, lifesaving blood transfusion should not be withheld. The patient's views should be explored at the earliest opportunity and documented.

Further Reading

- British Medical Association. (n.d.). *Children and Young People Toolkit: A Toolkit for Doctors*, available at: www.bma.org.uk.
- Care Quality Commission. (n.d.). "GP mythbuster 8: Gillick competency and Fraser guidelines", *GP Mythbuster 8: Gillick Competency and Fraser Guidelines*, available at: www.cqc.org.uk/guidance-providers/gps/gpmythbuster-8-gillick-competency-fraser-guidelines (accessed 31 August 2022).
- Great Britain. Department for Constitutional Affairs. (2007), *Mental Capacity Act 2005: Code of Practice*, TSO.
- Jane Titus S. (n.d.). Ethical guidelines for the anesthesia care of patients with Do-Not-Resuscitate orders or other directives that limit treatment committee of origin: Ethics (Approved by the House of Delegates, October 13, 1993). *American Society of Anesthesiologists*.
- Klein AA, Bailey CR, Charlton A, Lawson C, Nimmo AF, Payne S, Ruck Keene A, et al. Association of Anaesthetists: Anaesthesia and peri-operative care for Jehovah's Witnesses and patients who refuse blood. *Anaesthesia*. 2019; 74(1), 74–82.
- Milligan LJ, Bellamy MC. Anaesthesia and critical care of Jehovah's Witnesses, *Continuing Education in Anaesthesia Critical Care and Pain*. 2004; 4(2): 35–39.
- North Devon Health NHS. (n.d.). Consent Forms, *Patient Information Consent Forms*, available at: www.northdevonhealth.nhs.uk/patient-information/patient-information/consent/consent-forms/ (accessed 31 August 2022).

Critical Incidents

Ruth Doolin

What is a critical incident?

A critical incident is any preventable mishap, which leads to or could have led to an undesirable patient's outcome. A near miss is defined as an event with the potential to progress to the negative outcome if left.

Give some examples of critical incidents related to anaesthesia?

Examples of critical incidents affecting the cardiovascular system include cardiac arrest, myocardial infarction and arrhythmia. Affecting the respiratory system includes respiratory arrest, failed intubation, reintubation, airway injury, bronchospasm and aspiration. Neurologically, a critical incident could consist of convulsions or coning due to raised intracranial pressure.

Relating to regional anaesthesia, a high spinal block. Pharmacological critical incidents include adverse drug reaction, medication error or malignant hyperpyrexia.

Other critical incidents could include prolonged recovery stay, unplanned return to theatre, equipment failure or case cancellation.

What actions should be taken after a critical incident occurs?

It is important to document the incident in a legible, clear, and factual manner in the patient's notes. Patient records should be contemporaneous and easy to understand. In addition to documentation, there are several systems available to report critical incidents both locally and nationally, and these should be completed to allow learning to occur from the event. Where appropriate duty of candour guidelines should be followed and patients informed when something has gone wrong, an apology offered, appropriate support and a full explanation of the effects of what has happened.

What national and local reporting systems are used?

Incidents should be reported locally via risk management systems (e.g. DATIX), allowing for local analysis to get to the root cause of incidents, and learning how to prevent them occurring in the future. Presentation at local morbidity and mortality meetings can also help inform colleagues and allow discussion of events contributing to incidents. This works alongside the national NHS Reporting and Learning System (NRLS) which was first established 2001 by the National Patient Safety Agency (NPSA). This acts as the central database for all patient safety incident reports and is used to extract frequency and types of incidents.

Specific to anaesthesia, there is also the Safe Anaesthesia Liaison Group (SALG) which reviews and analyses anaesthesia-related serious incidents. Incidents can be reported directly, and also taken from those reported to the NRLS.

For specific incidents, such as drug reactions or blood product reactions there are separate schemes in place e.g. Medicines and Healthcare products Regulatory Agency (MHRA) yellow card scheme, and the Serious Adverse Blood Reactions and Events (SABRE) which can be used.

How is incident reporting used to ensure successful learning?

There are 4 key activities described in ensuring successful learning from incidents.

These include:

- **Data input** – which should be impartial and objective. Engagement from all healthcare professionals is vital. Incidents can be reported by all members of the multidisciplinary team.
- **The data** – reporting systems should allow plenty of ‘free text’ responses to allow a descriptive version of events to be reported. This better reflects the true nature of the incident, timings and highlighting relevant factors leading up to the event.
- **Analysis** – This requires breakdown of the data to find significant learning outcomes, and requires specialist expert analysis, preferably using a standardised methodology.
- **Feedback** – Lessons should be shared with all parties, with the goal of shared learning to improve patient safety, and avoidance of blame.

How is a critical incident analysed?

To allow understanding of how a critical incident has occurred, a detailed analysis of events in the lead up to the incident should be established, and subsequently used to identify interventions at different steps to prevent this happening again.

Psychological and human factors should be considered alongside organisational factors. The human factors approach focusses more on the pre-existing organisational factors which set up the conditions to allow the incident to occur.

‘The Swiss Cheese model’ of accident causation, described by James Reason in 1997, has been adapted to healthcare and is a common framework used for analysing incidents and identifying both active and latent conditions leading to failures.

The principle of this model is that safe systems have multiple layers of defences against incidents occurring, preventing a single point of weakness. However, none are perfect, and they deteriorate with time leaving ‘holes’ in the defence. With time and changing priorities of the organisation, sometimes these holes align subsequently allowing an incident to occur.

What is the difference between active and latent failures?

Human failure can be divided into errors or violations. These are examples of active failures. Error is the ‘failure of planned actions to achieve a desired goal’ and can be either ‘skill-based’ (unintended) or ‘mistake-based’ (intended action with unintended diagnostic error.) Skill-based errors can be called either slips (attention-based) or lapses (memory-based).

Mistakes occur where the actions go as planned; however, the plan was inadequate to achieve the desired outcome. Rule-based mistakes occur when people have ‘rules’ or

‘procedures’ for dealing with an event, and occur due to incorrect application of a good rule, application of a bad rule, or failure to apply the good rule. Knowledge-based mistakes occur when a problem has to be ‘worked out’ without use of pre-learned rules.

Conversely, violations are seen where there is a negative patient safety culture, and are intentional deviations from safe practice. These can be subdivided into routine, optimising or necessary violations. Routine violations includes ‘cutting corners’, optimising violations are performed to achieve personal goals as opposed to task-related goals, and necessary violations are seen as the only route to completing a task, where any rules or procedures are deemed inappropriate.

Latent failures relate to decisions taken by the organisation, which create the conditions for unsafe acts to occur e.g. staffing, workload, supervision, stress, communication, equipment, conflict of priorities. The effects of these failures may lay dormant for some time before the effects are realised.

What do you think are the barriers to reporting critical incidents?

The main barriers to reporting include fear of punishment or blame, legal implications and poor safety culture in an organisation. In addition to this, lack of understanding about what should be reported, how the information will be used and how patient safety can be improved as a result, can prevent engagement with incident reporting.

Further Reading

Agbamu P, Menkiti I, Ohuoba E, Desalu I.

Critical incidents and near misses during anesthesia: A prospective audit. *Journal of Clinical Sciences*. 2017; 14(1): 18.

GMC. GMC When things go wrong: Openness

and honesty when things go wrong: The professional duty of candour. 2015; available at: www.gmc-uk.org/ethical-guidance/ethical-guidance-for-doctors/candour—openness-and-honesty-when-things-go-wrong (accessed 16 August 2022).

Johnson CW, Botting RM. Using reason’s model of organisational accidents in formalising accident reports. *Cognition, Technology and Work*. 1999; 1(2): 107–118.

Mahajan RP. Critical incident reporting and learning. *British Journal of Anaesthesia*. 2010; 105(1): 69–75.

Medical Protection Society. An essential guide to medical records. *An Essential Guide to Medical Records*. 6 July 2020; available at:

www.medicalprotection.org/uk/articles/an-mps-essential-guide-to-medical-records (accessed 16 August 2022).

NRLS. National reporting and learning system, *NRLS Reporting*. n.d.; available at: <https://report.nrls.nhs.uk/nrlsreporting> (accessed 16 August 2022).

Reason J. Understanding adverse events: human factors. *Quality and Safety in Health Care*. 1995; 4(2): 80–89.

Reed S, Arnal D, Frank O, Gomez-Arnau JI, Hansen J, Lester O, Mikkelsen KL, *et al*. National critical incident reporting systems relevant to anaesthesia: A European survey. *British Journal of Anaesthesia*. 2014; 112(3): 546–555.

Royal College of Anaesthetists. Royal College of Anaesthetists: Core level training, critical incidents. *Critical Incidents*. 14 August 2019; available at: www.rcoa.ac.uk/documents/cct-anaesthetics-core-level-training/critical-incidents (accessed 16 August 2022).

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